Archives of Dermatology and Syphilology

VOLUME 45 JUNE 1942 NUMBER 6

COPYRIGHT, 1942, BY THE AMERICAN MEDICAL ASSOCIATION

DERMATITIS DUE TO THE BLOSSOM OF GREVILLEA BANKSII

A NEWLY RECOGNIZED AND COMMON ENTITY IN HAWAII

HARRY L. ARNOLD JR., M.D.

HONOLULU, TERRITORY OF HAWAII

The occasional production of dermatitis venenata by contact with flowering plants has been commented on briefly by several writers. Weber,[1] in an extensive list of cutaneous irritants, mentioned the following plants as occasional sensitizers: daffodil (Narcissus pseudonarcissus), buttercup (Ranunculus asiaticus), tree of heaven (Ailanthus altissima), gas plant ([Fraxinella] Dictamnus albus), olive spurge ([Mezereon] Daphne mezereum), catalpa (Catalpa sp.) and pasqueflower (Anemone patens). The list did not specify whether the blossoms or the leaves or both were irritating. Shelmire[2] discussed dermatitis from flowers and shrubs, remarking that florists and housewives, especially the latter, are naturally the most frequent sufferers from this. The most common skin sensitizers among the flowers and shrubs in his experience were the primose (Primula obconica), painted daisies ([Pyrethrum] Chrysanthemum sp.), feverfew (Chrysanthemum parthenium) and other members of the chrysanthemum family; less frequent offenders were the Shasta daisy, narcissus, tulip, iris, vinca, petunia, verbena, carnation, zinnia, cosmos, Amoor River privet and trumpet vine. He felt, however, that in all instances the responsible portions of the plants were the leaves and stems rather than the blossoms.[3] Osborne and Putnam[4] mentioned 10 florists found to be "sensitive to chrysanthemums"; no specific statement was made, however, regarding the division of responsibility between blossoms and

Read before the staff of The Clinic in August 1941.

1. Weber, L. F.: External Causes of Dermatitis: A List of Irritants, Arch. Dermat. & Syph. **35**:129 (Jan.) 1937.

2. Shelmire, B.: Contact Dermatitis from Vegetation, South. M. J. **33**:337 (April) 1940.

3. Shelmire, B.: Personal communications to the author.

4. Osborne, E. D., and Putnam, E. D.: Industrial Dermatoses, J. A. M. A. **99**:972 (Sept. 17) 1932.

leaves of the plant. They also recorded 1 instance of sensitivity to tulips, likewise in a florist; again, the effect of the leaf was not mentioned.

In a general way, however, little work has been done on the question of the limitation of sensitizing properties to certain portions of plants, and in particular to either the leaves or the blossoms. When such limitation has been demonstrated, it has appeared that the leaves, and not the flowers, contained the sensitizing principle. This has been shown to be true in at least certain cases of sensitivity to the primrose (Primula obconica),[5] reactions being obtained only with the leaves and not with the blossoms, though Shelmire[2] stated that the antigenic oleoresins "appear on the leaves, stems and flowers" of most dermatitis-producing plants. Shelmire[6] has shown for the poison ivy (Rhus toxicodendron or Rhus radicans) that the pollen does not contain the antigenic material found in the leaves; he did not state definitely, but seemed to imply, that the antigenic resin may be present in the blossoms. It has been demonstrated many times that the bark from both the twigs and the roots of the poison ivy is as rich in antigenic oleoresin as the leaves are.[7]

Brier[8] recently reported a case of contact sensitization to the "flowering parts"—and not to the leaves—of the thoroughwort (Eupatorium altissimum), with successful desensitization; however, it seems possible that portions of the stem were included in the "flowering parts," and, in any event, this was an isolated case. With this single possible exception, I have been unable to find any report of an instance of proved sensitization to the blossom of a plant and not to its leaves.

This paradoxic situation is true, however, of an ornamental plant found in Hawaii, which is known colloquially as the kahili flower (or, less often, the rain flower) and properly as Grevillea Banksii (figs. 1 and 2). Not only is its skin-sensitizing property confined (or very nearly so) to its blossom, but this property is extremely well developed, to such a degree that the plant has been the direct cause in more than three fourths of all the cases of dermatitis venenata of the plant type in my own practice. In a previous preliminary report several instances of this were described.[9]

---

5. Simpson, C. A.: Primrose Dermatitis and Its Relation to Anaphylaxis, J. A. M. A. **69**:95 (July 14) 1917.

6. Shelmire, B.: Contact Dermatitis from Weeds: Patch Testing with Their Oleoresins, J. A. M. A. **113**:1085 (Sept. 16) 1939.

7. (*a*) Krause, G. L., and Weidman, F. D.: Ivy Poisoning, J. A. M. A. **84**:1996 (June 27) 1925. (*b*) Shelmire, B.: The Nature of the Excitant in Poison Ivy Dermatitis, Arch. Dermat. & Syph. **42**:405 (Sept.) 1940.

8. Brier, A. J.: Contact Dermatitis from Thoroughwort (Eupatorium altissimum), J. Allergy **11**:402 (May) 1940.

9. Arnold, H. L., Jr.: "*Kahili* Flower" (Grevillea Banksii) Dermatitis, Hawaii M. J. **1**:15 (Sept.) 1941.

Fig. 1.—Grevillea Banksii.

UNIV. OF MICH.

This fact is the more remarkable because in Honolulu, and in Hawaii generally, the mango tree (Mangifera indica) has for many years been regarded as the only important dermatitis-producing plant (fig. 3); it has much the same reputation as have the poison ivy and poison oak in the

Fig. 2.—A typical kahili.

mainland United States. It is by no means unusual for a patient with an acute vesicular dermatitis to suggest the diagnosis of "mango rash" or for physicians to make an offhand diagnosis of "mango dermatitis" when confronted with such an eruption. The mango is, moreover, so abundantly distributed throughout the Territory of Hawaii that the question of opportunity for contact need never arise; there are at least half as

many mango trees in Honolulu as there are front yards. The fact that the mango is capable of producing dermatitis is not to be questioned for a moment; it seems likely, however, that it is by no means the most frequent cause of dermatitis venenata here. It shares with Rhus toxicodendron the firmly established but undoubtedly undeserved reputation of being able to produce dermatitis from a little distance or through the medium of smoke from a bonfire in which it is burning. It also has other interesting, if not properly established, properties; for example, it is said that many persons are affected by the skin of the unripe fruit but not by that of the ripe fruit, whereas others are reactive only to the leaves and not to the skin of the fruit or only to the sap and not to the leaves or fruit; and—at the opposite extreme—some persons believe that even

Fig. 3.—Young mango tree.

eating the peeled fruit will give them dermatitis. The latter observation seems the most likely to be fallacious; all of them, however, would merit investigation.

BOTANICAL CONSIDERATIONS

The genus Mangifera, like the genus Rhus, belongs to the family Anacardiaceae. So does the cashew (Anacardium occidentale), the rind of the nut of which is said to contain an extremely potent skin-sensitizing (or at least not universally irritating) substance. This tree is also grown in Hawaii, though it is not abundant. The comocladias (Comocladia spp.), anacardiaceous trees mentioned by Pardo-Castello [10]

10. Pardo-Castello, V.: Dermatitis Venenata: A Study of the Tropical Plants Producing Dermatitis, Arch. Dermat. & Syph. **7**:81 (Jan.) 1923.

as widespread and frequent skin sensitizers in the West Indies, are not found here. The genus Rhus is represented in Hawaii, though it has such limited distribution that no practical importance can be attached to its presence. Rhus toxicodendron does not occur here at all. There is an indigenous Hawaiian Rhus [11] (fig. 4), species semialata (Murray), variety Sandwicensis (Engler), only a few specimens of which are known to exist on this island, two in the Manoa arboretum of the Hawaii

Fig. 4.—Single leaf of Rhus semialata (one-third size).

Sugar Planters' Association experiment station and several in upper Nuuanu Valley. It grows on the island of Hawaii, along the Hamakua coast, as a roadside weed. The Hawaiians know it as ***neneleau*** or ***neleau.*** In the same arboretum there are specimens of Rhus insignis, Rhus succedanea (the Japanese wax-tree) (fig. 5) and two unidentified species of Rhus which have been imported from Japan and from Samoa. All of

11. Rock, J. F.: The Indigenous Trees of the Hawaiian Islands, Honolulu, The Author, 1913, p. 262.

these are more properly grouped with the sumacs than with the "oaks and ivies"; and their skin-sensitizing properties have not been investigated, though Bailey [12] stated that Rhus succedanea has the reputation of being poisonous. The nosologic relations of the plants mentioned are shown in the accompanying tabulation (from Bailey,[12] slightly modified after Shelmire):

Division IV Phanerogamia
- Subdivision II Angiospermae
  - Class II Dicotyledoneae
    - Subclass I Archichlamydeae (Choripetalae and Apetalae)
      - Order 29 Proteales
        - Family: Proteaceae
          - Genus: Grevillea
            - Species: Banksii (kahili flower)
            - robusta (Australian silk oak)
            - Thelemanniana
      - Order 39 Sapindales
        - Family: Anacardiaceae
          - Genus: Mangifera
            - Species: indica (common mango)
          - Genus: Rhus
            - Species: radicans (toxicodendron), quercifolia, diversiloba, microcarpa, Rydbergii (poison ivy or oak)
            - vernix (poison sumac)
            - verniciflua (lacquer tree)
            - succedanea (wax tree)
            - semialata var. Sandwicensis (*neneleau*)
          - Genus: Comocladia spp.
          - Genus: Anacardium
            - Species: occidentale (cashew)
    - Subclass II Metachlamydeae (Sympetalae)
      - Order 47 Primulales
        - Family: Primulaceae
          - Genus: Primula
            - Species: obconica (primrose)

12. Bailey, L. H.: The Standard Cyclopedia of Horticulture, New York, The Macmillan Company, 1937.

Grevillea Banksii (Degener [13] called it Stylurus Banksii for the sake of historical accuracy) is a member of a large genus, all the members of which are indigenous to Australia, chiefly Queensland. Its common name in Hawaii, kahili flower, comes from the close resemblance of its racemes to the ancient Hawaiian ceremonial kahili (fig. 2), a long staff topped by a cylindric cluster of radiating feathers, somewhat after the fashion of a huge test tube brush with coarse rather sparse bristles. This name is really more properly applied to Hedychium gardnerianum, the kahili ginger, a much larger flower shaped somewhat like a kahili but

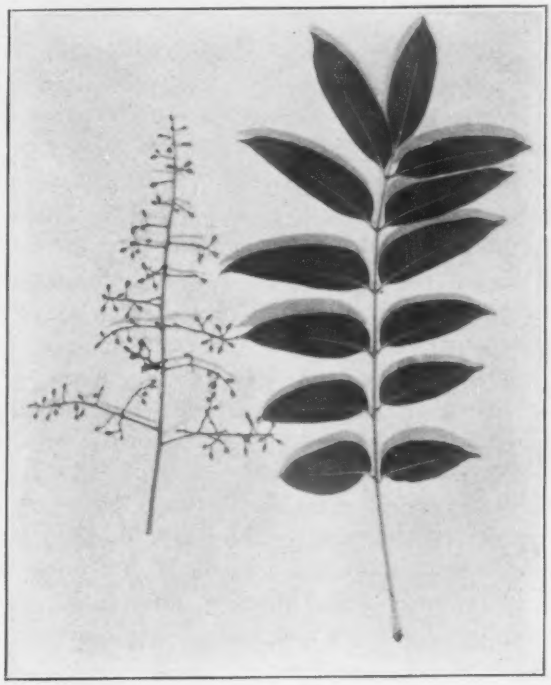


Fig. 5.—Leaf and immature blossom of Rhus succedanea (one-third size).

entirely unrelated to the Grevilleas. Grevillea Banksii was brought to Hawaii from Australia at least forty years ago,[14] perhaps more, and is now fairly widespread here, though its disinclination to grow in Hawaii at elevations below about 300 feet has limited its spread somewhat. Thus it is found not at all in the city of Honolulu but only in the higher portions of the valley residential districts, a few isolated trees being found in the upper part of Nuuanu valley and several small commercial orchards

13. Degener, O.: Flora Hawaiiensis, Honolulu, The Author, 1932.

14. McGuire, T. (Forest Ranger, Board of Agriculture and Forestry, Territory of Hawaii): Personal communication to the author.

in the upper end of Manoa valley. A professional botanist, a resident for many years, recently informed me in confidence that he had never yet seen it growing! This observation is certainly the experience of most persons; the cut racemes, which are showy and inexpensive and therefore often incorporated into bouquets, are apparently the only significant source of contact with the plant. Their use is so widespread among local florists that a history of contact—as with the mango—should probably not be regarded as essential in arriving at an etiologic diagnosis. One man, who presented himself with streaks and patches of tense vesicles on the flexor aspect of his right wrist, stated that he had not been in the vicinity of kahili flowers; however, after the incriminating results of patch tests and further questioning, he recalled having passed cigarets across a dinner table centerpiece which contained kahili flowers. On the island of Hawaii I have seen lei (wreath) vendors at the airport and at the steamship wharf offering leis of kahili flowers for sale. This well illustrates the fact that they have as yet no widespread reputation as a possible source of dermatitis; one would hardly expect to find wreaths of poison ivy offered at 50 cents apiece for wear about the neck!

Grevillea Banksii (fig. 6) is a shrub or low-growing compact tree attaining an average height of only 8 to 10 feet (245 to 300 cm.) in Hawaii, though Australian specimens are said to grow to twice this height. Its leaves are dark green above, silvery below, 4 to 8 inches (10 to 20 cm.) long and deeply pinnatifid, with lanceolate segments. The racemes are terminal and erect and 2 to 4 inches (5 to 10 cm.) long; there are two varieties, one red and one pale yellow. The flowers composing the racemes are small (less than 1 inch [2.5 cm.] long), perfect, tomentose (covered with minute hairs) outside and apetalous; the recurving calyx is split down one side; the style is long and curved, with its tip recurved and attached to the calyx until the flower is mature, when it is released and springs out nearly straight. The anthers are borne on the sepals.[13]

There is only one other Grevillea in Hawaii, species robusta, commonly known as the silk or silky oak, silver oak, he oak or Australian oak (fig. 7). It is much more widely distributed throughout the islands than Grevillea Banksii and differs from it in several respects. It is a large tree, said to attain a height of a hundred feet (3,000 cm.) or more in Australia, though Hawaiian specimens are in general no more than half this height, with trunks a foot or 2 (30 to 60 cm.) in diameter. The leaves are twice pinnatifid, the broader segments being divided once or twice again nearly to the midrib, generally on the outer side only. The flowers, smaller and orange-yellow, are borne in terminal and axillary horizontal racemes, singly or several together. Grevillea robusta also grows in California and Florida, as well as in its native Australia. Its

wood, which is exceedingly tough and durable, has been used for barrel staves and furniture.

A third Grevillea, species Thelemanniana, is said by Bailey [12] to be a popular shrub in California; it bears pink flowers with green tips, arranged in racemes rather smaller than those of Grevillea Banksii. No further information is available regarding this plant.

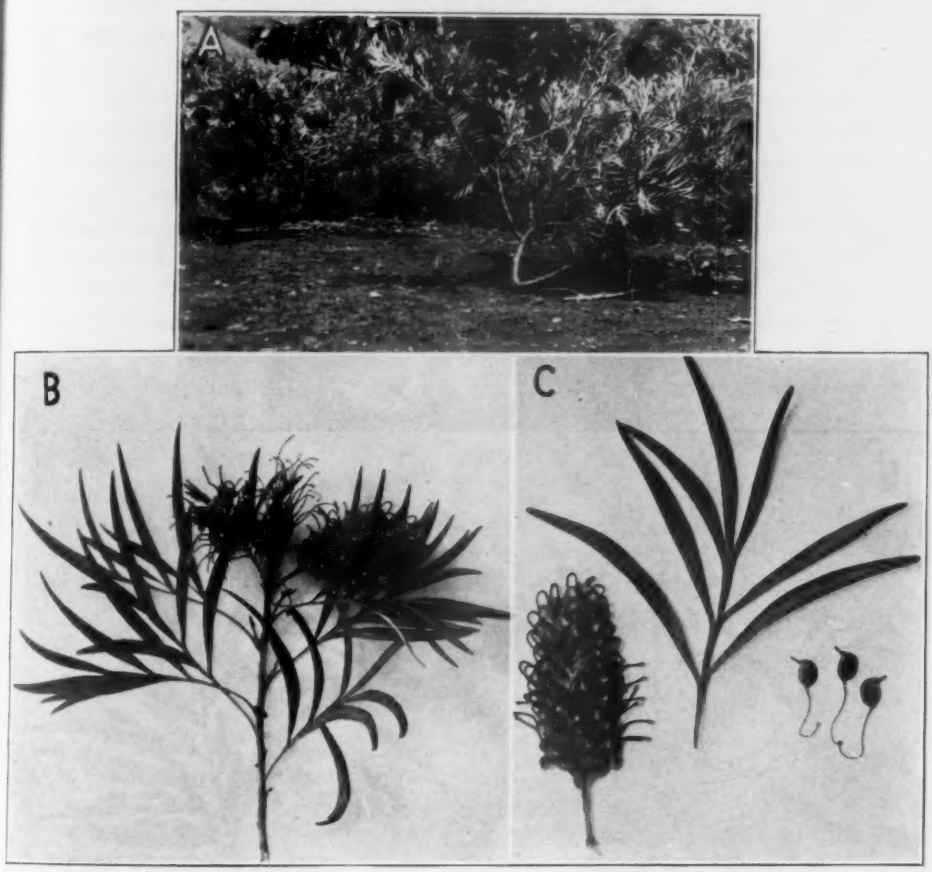


Fig. 6.—Grevillea Banksii. *A*, shrubs about a year old. *B*, branch showing erect terminal racemes, one of which has ripened to form seeds (one-seventh size). *C*, raceme, leaf and seeds (one-fourth size).

CLINICAL CONSIDERATIONS

As observed by Shelmire in his cases of dermatitis from flowering plants,[2] women are affected by kahili flowers far more often than men (there was a ratio of 6 to 1 in my series); the usual story is that they arranged or at least touched a bouquet which contained the offending blossoms. The incubation period is ordinarily about three days; two

days is not particularly unusual, but no case was observed in which the contact was known to have been limited to twenty-four hours before the onset of the eruption.

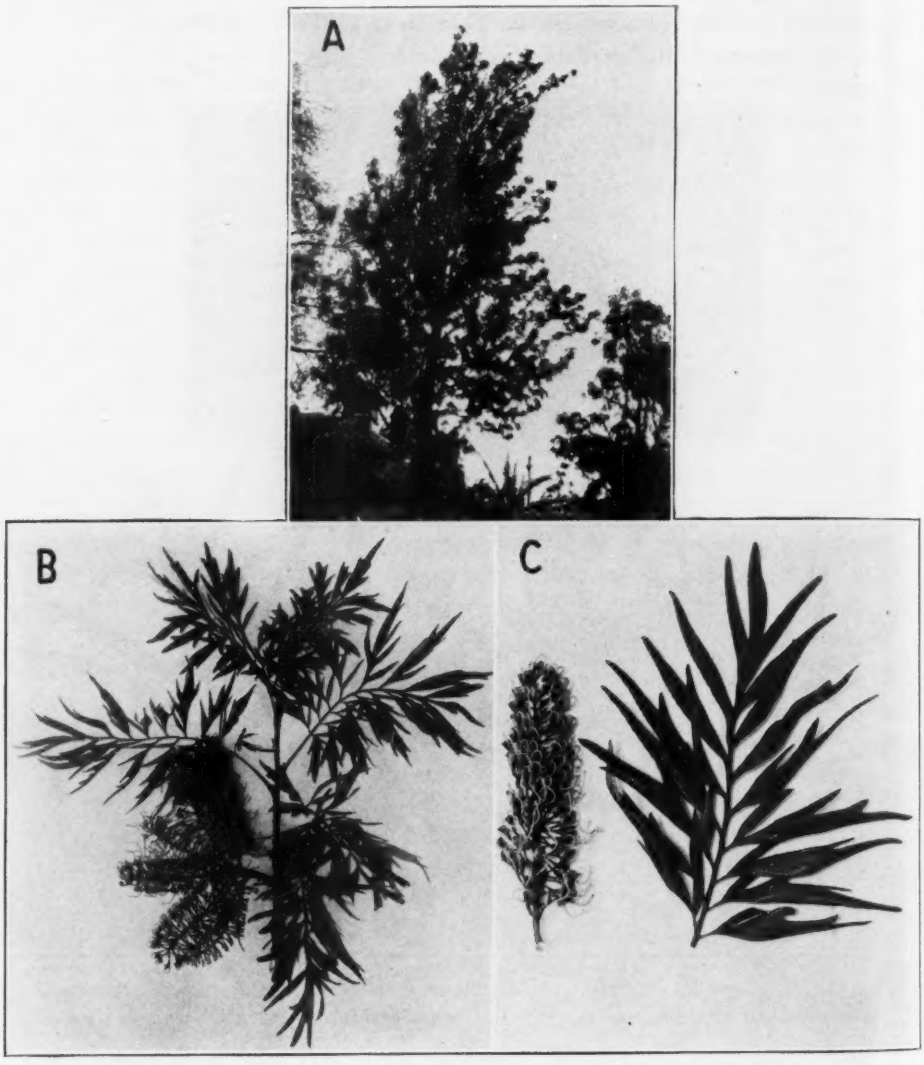


Fig. 7.—Grevillea robusta. *A*, medium-sized specimen. *B*, branch showing grouped horizontal racemes (one-seventh size). *C*, single raceme and leaf (one-fourth size).

In many cases the earliest symptom is itching and redness of the eyelids and ears; in about a like number, however, tense clear pruritic vesicles, 1 or 2 mm. in diameter, first appear on the wrists and backs

of the hands, frequently associated with, or followed shortly by, redness on the face and neck. This is probably explained by the initial deposition of the irritant material in liberal amounts in sharply localized areas on the hands; these areas are likely not to be rubbed much for a time and therefore become vesicular rather early. Material deposited on the palmar surfaces of the hands, however, usually produces no local irritation but is transferred by direct contact to the face and neck, where it is spread out in a relatively thin layer sufficiently concentrated to produce not vesiculation but only redness. In 1 case, however, a nurse was affected as a result of putting her face directly against the blossoms; several oval patches of severe vesicular dermatitis developed on the cheeks and forehead and only a few trivial lesions on the forearms and hands. Lesions about the lips, which are frequently observed in cases of dermatitis due to oleoresin in the skin of the mango, do not of course occur with any regularity in dermatitis due to kahili flowers. Aside from these minor variations, however, there is no feature of this condition that is basically different from the dermatitis produced by any other skin-sensitizing plant; and indeed patients who have previously suffered from poison ivy dermatitis have been heard to remark that the eruption from the kahili flower is "exactly the same."

INVESTIGATIVE MEASURES

Proof that the blossom of Grevillea Banksii was responsible for acute contact dermatitis in a majority of the cases here recorded rests in most instances on a strongly positive cutaneous reaction to the application of an ether extract of the dried blossoms prepared according to the technic developed and reported by Shelmire.[6]

To recapitulate briefly, this consists in drying the leaves or blossoms in a dark room (to prevent fading), separating the leaves or flowers from the twigs and stalks and rubbing them between the palms to a finely divided state (instead of using a grinder; Shelmire[3] has similarly modified his own original technic), placing the material in a pint or quart jar with a screw top and covering with commercial sulfuric ether. The ether is decanted after twenty-four hours and allowed to evaporate (preferably on a thermostatically controlled hotplate, to prevent cooling and consequent accumulation of dew); the sticky residue is then weighed (most easily determined by finding the difference between the weight of the beaker containing the material and that of the originally empty beaker) and redissolved in ether, usually to make a 10 per cent (weight-volume) solution. This is placed in ordinary homeopathic vials.

Tests are made by inverting the vial so as to wet the cork, then removing the cork and applying its moist end to the patient's skin, where the ether solution quickly evaporates, leaving a deposit of colored material to mark the test site. For multiple tests the back is the site of choice; for six or eight tests or less, the flexor surfaces of the forearms will serve well and are far more convenient. All tests herein reported were made on the arms.

Twenty-six patients of 31 tested gave a positive reaction to this test, as shown in the accompanying table.

All of these 26 patients were likewise tested with an extract of the leaves of Grevillea Banksii; only 7 manifested any reaction, and in all of these it was slight. Twelve of the 26 were tested not only with the extracts of the blossoms and leaves but also with the blossoms and leaves

Results of Contact Tests with Leaves and Flowers (or Extracts Thereof) of Grevillea Banksii, Grevillea Robusta and Mango

				Grevillea Banksii				Grevillea Robusta	
No.	Sex	Contact	Mango Leaf Extract	Flower Extract	Flower	Leaf Extract	Leaf	Flower Extract	Flower
1	F	G. Banksii flower	—	++++	..	—	..	..	..
2	F	—	—	+++	..	+	..	..	..
3	F	G. Banksii flower	—	++++	..	—	..	..	..
4	M	—	—	++++	..	—	..	..	..
5	M	G. Banksii flower	—	+++	..	—	..	..	..
6	F	Mango	—	++++	..	+	..	..	..
7	F	G. Banksii flower	—	++++	..	—	..	..	..
8	F	—	—	+++	..	—	..	..	..
9	F	—	—	+++	..	—	..	..	..
10	F	G. Banksii flower	—	+++	++++	—	—	..	..
11	M	G. Banksii flower	—	++++	..	+	..	..	..
12	F	—	—	+++	..	+	..	..	..
13	F	—	—	++++	++++	—	—	..	..
14	F	G. Banksii flower	—	++++	..	—	..	..	..
15	F	G. Banksii flower	—	++++	..	—	..	..	..
16	M	Mango	++++	—	..	—	..	..	..
17	M	Mango	++++	—	..	..	..	..	..
18	F	G. Banksii flower	—	++	++	—	—	—	..
19	F	G. Banksii flower	—	++++	++++	—	—	—	..
20	F	Mango	+++	—	—	—	—	—	..
21	M	Mango	—	—	—	—	—	—	..
22	F	Mango	++	++++	+++	—	—	—	..
23	F	—	—	—	—	—	—	—	..
24	F	G. Banksii flower	+	++++	++++	+	—	—	..
25	F	Mango	—	—	—	—	—	—	..
26	F	G. Banksii flower	—	+++	++++	—	—	—	..
27	M	Mango	+++	++	++++	—	—	—	..
28	M	—	—	+++	+++	—	+	—	..
29	F	—	—	++++	+++	+	—	—	..
30	F	G. Banksii flower	—	+++	+++	—	++	—	..
31	F	G. Banksii flower	—	++++	++	+	+	—	..
		Controls							
10	..	Normal	—	—	..	—	..	..	..
16	..	Dermatitis venenata	—	—	..	—	..	..	..
6	..	Normal	..	..	—	..	—	—	— (4 cases)

themselves; with the leaves, an ordinary twenty-four hour covered patch test was used in all cases; with the blossom, however, this produced overly severe reactions, and many tests were made by merely rubbing a small area of skin with the style of the flower and then covering the site. All the persons sensitive to blossom extract proved highly sensitive to the blossom itself; only 1 of the 3 patients sensitive to leaf extract reacted to the leaf itself, while 2 others did not. Ten normal persons

manifested no reaction to the extracts of either blossom or leaf, and this was likewise true of 16 patients with dermatitis venenata of other than plant type; 6 healthy persons were given twenty-four hour covered patch tests with the blossom and leaf of Grevillea Banksii and manifested no reaction except that 2 showed slight sharply localized primary irritation which seemed to be produced by the fine hairs on the tomentose outer surface of the calyx. The style of the flower was used thereafter, and no such reactions were observed.

Because the blossoms of Grevillea Banksii may be either deep red or pale yellow, flowers of the two colors were at first extracted separately, and patients were tested with each. After 5 instances of identical reactions to the two, however, this procedure was discontinued, and a mixture of the blossoms was employed in all subsequent tests. It was found similarly that the red and white varieties produced perfectly comparable reactions when covered patch tests were made with the blossoms themselves.

On one occasion (in case 10) a single flower was carefully divided into style, anther and calyx, and covered patch tests were made with each part. There was no reaction to any at the end of twenty-four hours, when the patches were removed; at forty-eight hours there was erythema at each test site, and at seventy-two hours there was an equally severe vesicular reaction at each test site. No reaction occurred in this patient to a twenty-four hour covered patch test with the bruised fresh leaf. Reactions to the extracts were consistent.

It is of course recognized as a fundamental shortcoming of the patch test that like all similar tests it usually bears no direct relation to the presence of a dermatitis. All that these tests establish is the fact that 26 persons suffering from acute vesicular contact dermatitis were also highly sensitive to—and might well have been so affected by—the blossom (and not the leaf) of Grevillea Banksii. The history of contact lends a little weight of course, more in some cases than in others. Of these 26, only 13 recalled having been in the immediate vicinity of kahili flowers within a few days prior to the onset of their eruption. Five gave a history of close contact with mango leaves or fruit or both; the remaining 8 could recall no contact which seemed at all significant.

Two of the 26 patients, however, happened to come under observation at a time when their eruption was beginning to subside, and they manifested a phenomenon which provided strongly suggestive evidence of the specificity of the test: a striking aggravation of the healing cutaneous lesions within twenty-four hours of the time of application of the test materials, followed in about thirty-six hours by strongly positive reactions to the blossom and blossom extract and negative reactions to the other test substances.

Three of the 26 persons manifested some sensitivity to the mango leaf extract as well; in 2 this was much less than their apparent reactivity to the kahili flower extract, but in 1 it was slightly more. The last-mentioned person might therefore, so far as the patch test was concerned, have had a "mango rash"; it happened that there was a history of mango contact only, and so it would probably be best to group this patient with the 3 who gave a strongly positive reaction to mango leaf extract and a negative reaction to the Grevillea Banksii test materials. If this is done, the list of 31 subjects would then include 4 with dermatitis venenata due to mango leaf and 25 with dermatitis venenata due to Grevillea Banksii blossoms, giving a ratio of approximately 6:1. It should actually be 8:1, for 1 of the 4 was not actually a patient but a physician who inadvertently "tested" himself by contaminating his finger tips in the course of using the extracts to test a patient in the usual manner. Unconscious transfer of the material to his face in the course of the day resulted in a dermatitis so severe as to incapacitate him completely for four days. He subsequently recalled having suffered from dermatitis brought on by contact with mangoes on several previous occasions.

Fourteen of the 31 tested persons were also tested with an ether extract of the blossoms of Grevillea robusta; no reactions were observed. Twenty-four hour covered patch tests with the flowers themselves were made on 4 controls, but no reaction was manifested.

Shelmire has shown [7b] that the active principle in the production of dermatitis by the poison ivy is a urine-soluble fraction of the oleoresin of the plant. An attempt was made to duplicate this procedure for Grevillea Banksii blossoms, but without success. Six persons, all of whom were highly sensitive to the ether extract of these blossoms, failed to react at all to a 10 per cent ether solution of material extracted from the dried flowers by digestion with fresh urine, according to Shelmire's method.

SUMMARY AND CONCLUSIONS

Grevillea Banksii, a decorative proteaceous plant, is the most frequent cause of dermatitis venenata of the plant type in dermatologic practice in Honolulu.

This plant is remarkable in that its antigenic material is found only in the blossoms and not, as is the case with nearly all other dermatitis-producing plants, in the leaves.

The antigenic material appears to be present in comparable amounts in the calyx, anthers and style of both the red and the yellow varieties of blossom.

The antigenic substance appears to differ from that of Rhus toxicodendron in that it cannot be extracted from the plant by digestion with urine and from the urine solution by digestion with ether.

Cross sensitization to the blossoms of a closely related tree, Grevillea robusta, does not appear to occur.

NOTE.—Dried leaves of Rhus semialata and Rhus succedanea were sent to Dr. Bedford Shelmire, who prepared ether extracts of them, with which he tested several volunteers who were sensitive to poison ivy. None reacted to the extract of Rhus semialata, but all reacted strongly to a significantly high dilution of the extract of Rhus succedanea. This observation, together with the fundamental botanical similarities of the two plants (the Japanese wax tree and the poison ivy), suggests that more widespread planting of the former might well lead to the introduction of true poison ivy dermatitis into Hawaii.

Edward L. Caum, botanist with the Hawaii Sugar Planters' Association experiment station, made possible the collection of the specimens of Rhus and assisted in the preparation of the technical botanical sections of the paper.

881 Young Street.

DERMATITIS DUE TO A TRANSPARENT ADHESIVE TAPE

HARRY KEIL, M.D.
NEW YORK
AND
EUGENE S. BERESTON, M.D.
BALTIMORE

The application of various adhesive plasters to the skin may occasionally produce irritant effects, which are generally classified in two separate categories: (1) a simple traumatic type, attributed to or influenced by many mechanical factors, such as undue tackiness or creasing at the site of application, and (2) a variety due to true hypersensitivity to one or more ingredients in the particular product. As a rule, these reactions can be easily differentiated; the former is usually manifested as a simple transitory irritation of the skin, whereas the latter shows the clinical features of a more persistent, genuine contact eczema (dermatitis). This distinction cannot always be made on the basis of clinical inspection alone. Nevertheless, in the vast majority of instances the differentiation is fundamental. The mechanical type is so commonly encountered, especially when adhesive tapes are left on the skin for any length of time, that it may be regarded as banal. This reaction has probably little practical importance, except in cases in which tapes are retained in place for many days (as in strappings for a fracture), and it can be eliminated in large part, but not always, by paying attention to mechanical factors, such as avoiding creases. On the other hand, certain other mechanical properties may be influenced considerably by chemical constituents in the product, for example those which promote undue tackiness. It is probable that no tape on the market is entirely free from this usually minor inconvenience, though at times some seem to be mechanically more irritating than others.

In contrast to the reactions just described, true hypersensitivity to a particular substance, usually some form of rosin, or to combinations of substances, with the production of a contact eczema is relatively uncommon in the average person. The term dermatitis is best limited to this type of reaction, if confusion is to be avoided. There is no adhesive tape which can be said to be absolutely free from this complication, even

From the Department of Dermatology, service of Dr. F. Wise, Montefiore Hospital for Chronic Diseases, New York.

though manufacturers have made laudable efforts to eliminate from their formulas those substances more readily capable of causing this reaction.

There is a third type of irritant effect, which is extremely rare. This is featured by a primary toxic action on the epidermis resembling an alkali burn, with dissolution and fairly deep necrosis of this layer. Stauffer [1] mentioned 2 cases of this phenomenon, but he was unable to investigate them. The condition in such instances may be due possibly to the inclusion of substances capable of acting as primary irritants, such as those found in some specimens of turpentine. This aspect requires further study.

Finally, rare cases have been described in which the application of adhesive plaster was accompanied by a more or less generalized eruption, with many lesions occurring at points far removed from the original site of application. This phase of the subject is somewhat involved and will not be discussed here except to note that such reactions are probably due to the absorption of substances incorporated in the adhesive material.

The data bearing on the first two phenomena, the most important ones to be considered, apply generally to the various adhesive tapes made in this country, especially those based on essentially similar formulas. However, these rules will undoubtedly be violated as newly discovered substances are used. The conclusions drawn by some investigators regarding desensitization to "the ingredients of adhesive plaster" and the apparently paradoxic observation of patients intolerant to a particular kind of adhesive tape on one occasion yet tolerant at another time need to be evaluated in the light of the knowledge that changes in formula are occasionally made by the manufacturer and that such information is not always available.

Transparent adhesive tape has recently come into general usage, particularly among ophthalmologists, and the advantages of this product have been noted by a number of observers.[2] These advantages include, among others, a probable lesser incidence of ordinary traumatic irritation to the skin under the conditions to which this type of adhesive tape is usually subjected. In general, our observations tend to substantiate this, but we are unable to accept the implication that there is absence of true hypersensitivity to the complex mixture of ingredients that go into the manufacture of this tape or, for that matter, any other of the numerous adhesive plasters on the market. Among ophthalmologists

1. Stauffer, H.: Die Ekzemproben, Arch. f. Dermat. u. Syph. **162**:517, 1931.

2. (*a*) Bogart, D.: Use of Cellulose Adhesive Tape for Retaining Dressings for the Eye, Arch. Ophth. **17**:944 (May) 1937. (*b*) Wilson, C.: A Better Adhesive Tape for Use After Surgical Treatment of the Eye, ibid. **25**:334 (Feb.) 1941.

who have extensively used this product, Bogart [2a] encountered 5 instances of "dermatitis" in a mild form among 2,500 patients, and Hughes [3] also saw at least 1 example of this. However, there is no way of judging precisely the type of cutaneous alterations seen by these observers, nor were they able to investigate the nature of the causative agent.

Consideration of the data in this report will show why even transparent tape may occasionally produce a contact eczema in a susceptible person. From the point of view of the cooperating manufacturer, it is generally insufficient to point out that a dermatitis is due to an ingredient, especially if that substance is in reality a complex compound and particularly if it is a rare cause of the complication. A more adequate solution of the problem requires making patch tests with fractions of the implicated ingredient, based on a knowledge of the physical chemistry of the various substances in the formula and their relation to one another. It is likely that the precise cause or causes vary from person to person, and the efforts of the physician will be directed toward determining statistically the most frequent offender or combinations of offenders. Even under favorable conditions it may be difficult to arrive at precise conclusions owing to a number of causes: (1) the inherent peculiarities of the skin, the reactions of which may possibly be modified from time to time, chiefly by imperfectly understood nonspecific factors; (2) the possibility of group reactions to similar substances, as well as multiple, independent sensitivities, and (3) the difficulties, when one is using the separate ingredients for patch tests, in approximating their actual physicochemical state in the commercial product.

REPORT OF A CASE

I. C., a woman 50 years old, had an operation on the left eye for glaucoma. The postoperative dressing was held in place by transparent adhesive tape, which was reapplied for the next two weeks with each change of dressing. A few days after the operation the integument on the left side of the forehead and in the left malar region, to which the tape had been anchored, became the sites of a patchy erythematous and pruritic eruption. About two weeks later we saw the patient for the first time, and on this occasion the skin in the left malar region showed a scaly erythematous patch and that on the left side of the forehead a faint erythema. The diagnosis of contact dermatitis was evident. There was a previous history of sensitivity to other adhesives, but the patient was vague about the details.

A patch test with the tape applied to the forearm caused a bright erythema in twenty-four hours and superimposed vesiculation the following day, whereas the brand of adhesive which covered the site of the patch test failed to produce any alterations in the skin (fig. 1). The positive reaction became still more intense subsequently and required about ten days for regression.

---

3. Hughes, W. L.: Personal communication to the authors, Jan. 24, 1941.

Being unfamiliar with the ingredients in the transparent tape, we made patch tests with those substances commonly reported in connection with dermatitis due to adhesive plaster, namely, zinc oxide, dammar resin, orris and lead oleate. Of these only the lead oleate caused an erythematovesicular reaction within forty-eight hours. At the same time the transparent tape had been reapplied under a different brand of adhesive tape (so-called patch tape adhesive), and this time the entire site of the patch test, including the area covered by the latter adhesive, showed a pronounced vesicular reaction, so that the boundaries of the two plasters were indistinguishable.

On receipt of more detailed information from Dr. Louis Schwartz, of the Public Health Service, we made additional patch tests, using casein, liquid latex (slightly ammoniacal) and gum rosin (WW grade). These chemicals were obtained from a local concern. In order to avoid confusing reactions from adhesive tape itself, the substances were moistened and placed under gauze,

Fig. 1.—An intensely positive reaction elicited by transparent adhesive tape (lower arrow). The other brand of adhesive tape gave a negative reaction (upper arrow). Patch tests were read twenty-four hours after application.

and the edges were sealed with gum mastic. Of these only the gum rosin produced an erythematovesicular reaction, which became more pronounced in the next few days after removal of the patches.

Through the cooperation of the manufacturer samples of the actual ingredients of the transparent tape were furnished. These consisted of cellophane, two types of nonimpregnated rubber, a liquid hydrocarbon of the paraffin series, an ester gum (glycerinated wood rosin) and an antioxidant (a condensation product of a ketone and an aryl amine). In its final form the product has a p_H of about 7. The ester gum has an acid number of from 6 to 8; that is to say, it takes from 6 to 8 mg. of potassium hydroxide to neutralize the free acid in 1 Gm. of the ester gum. Wood rosin itself has an acid number of from 154 to 168. In other

words, practically all the abietic acid composing wood rosin has been combined with the glyceryl radical, with little free acid remaining in the ester gum.

Samples of the actual ingredients of the tape were applied under gauze sealed at the edges with collodion (an alcoholic ether solution of pyroxylin). Aside from moistening of the solids, the ingredients were used undiluted. Within twenty-four hours the sites of the ester gum showed an intensely pruritic vesicular erythema (fig. 2), and three days later the reaction was even more pronounced.

Fig. 2.—The upper arrow shows a positive reaction to the glycerinated ester gum. The lower arrow indicates the false positive reaction to the undiluted liquid hydrocarbon of the paraffin series (primary irritant effect). Patch tests were read twenty-four hours after application to the skin. The exfoliating appearance about the patches is due to the peeling of the collodion used to ring the patches.

The remaining substances caused no cutaneous changes, with the exception of the liquid hydrocarbon, which produced a dull erythema in twenty-four hours and a more pronounced erythema three days later. The latter reaction was simply a primary irritant (toxic) effect due to the use of the undiluted fluid, for subsequent dilution with 50 per cent liquid petrolatum caused no cutaneous changes, the results being controlled in a dozen normal subjects, including ourselves. Consequently, the only significant cutaneous reaction in our patient was that produced by the ester gum.

The major ingredient of commercial rosin is abietic acid, and this is especially true of wood rosin. Some investigators believe that abietic acid is the cause of the dermatitis in persons sensitive to ester gum or to plain rosin. We therefore obtained so-called crude abietic acid, a yellow-brown, crumbly, amorphous substance derived from gum rosin by acidification and high temperature. This substance seemed to correspond to the abietic acid described by Schwartz and Tulipan.[4] Actually, however, the product we obtained was a crude mixture of various acids, of which abietic acid was the chief ingredient, and, judging on the basis of its appearance, it was partially oxidized. This substance gave all the usual chemical tests for rosin, based on its content of abietic acid. After the completion of our investigation we obtained a partially purified abietic acid derived from wood rosin, and this was a pure white crystalline substance.[5] At this time, also, we did not know that the ester gum in the transparent tape was a glycerinated wood rosin. Nevertheless, the tests with the crude abietic acid permitted conclusions in respect to abietic acid and its companion acids as well as to whatever oxidized products were contained in the crude product.

Before testing our patient with the crude material, we applied it in the following forms to the skins of a dozen control subjects, including ourselves: as a dry powder, moistened in physiologic solution of sodium chloride and dissolved in ether. The results in all the controls were essentially negative, except for 1 person whose skin showed a slightly positive reaction to the moistened crude abietic acid and a questionable positive reaction to the dry powder. About a week later the skins of the control subject were scratched with the powder in the conventional manner, and again the results were negative. From this we concluded that crude abietic acid used in the form of powder or moistened or even dissolved in ether was not a primary irritant and probably not a frequent sensitizer.

The patient was then given patch tests with the crude abietic acid applied in the following ways: as a dry powder, moistened with water, moistened with physiologic solution of sodium chloride and dissolved in ether. None of these produced cutaneous changes, except for the ether solution. This change was obviously a false positive reaction due to the primary iritant effect of the solvent, the reaction being visible only at the edges of the applied square of gauze, where the ether had apparently attained its highest concentration. In an effort to see whether the ester gum contained a water-soluble fraction responsible for the dermatitis, we applied a patch with an aqueous filtrate of the substance immersed in water for some time (ester gum is practically insoluble in water); the result

4. Schwartz, L., and Tulipan, L.: A Text-Book of Occupational Diseases of the Skin, Philadelphia, Lea & Febiger, 1939.

5. Furnished by the Hercules Powder Co. through the courtesy of Dr. Lemuel C. McGee.

of this test was also negative. From these results we concluded that (1) the patient was sensitive to the ester gum (possibly this was due to some unknown fraction of it which, however, could not be extracted with water) and (2) the causative agent was probably not the abietic acid which was present in considerable amounts in crude abietic acid nor the ether or aqueous fractions of the crude substance.

The next step was to investigate the presence of humoral antibodies in the blood, according to the Prausnitz-Küstner technic, which we modified somewhat to suit the conditions of our study. Serum from the patient was collected, and 0.1 cc. was injected intracutaneously into a number of sites in the forearms of 2 subjects who had been found previously not to be sensitive to transparent tape. One day later crude abietic acid and the ester gum were scratched into two of the aforementioned sites and the transparent tape applied against the skin at a third site, together with controls. The results of all the tests were negative. We therefore concluded that humoral antibodies could not be demonstrated by the technic employed. We were unable to investigate the possibility of local antibodies in the skin itself.

At this time we did not know the original source of the ester gum, and, suspecting the possibility of turpentine as a sensitizer in our patient, we applied gum turpentine in 50 per cent dilution in olive oil to her skin. The result was negative. We now know that in view of the method of manufacture of this product it contains no turpentine.

Further investigations were prevented by the death of the patient from an intercurrent thrombosis of the coronary artery.

COMMENT

1. *Introduction to the Chemistry of Rosins.*—Rosin, or colophony, is the most important natural resin. It typifies the class of acid resins, owing to its high content of abietic and other acids. Two main types occur, depending on the source: gum rosin and wood rosin.

Gum rosin is derived from crude spirits of turpentine, which is an oleoresinous excretion of certain pine trees. By steam distillation the oleoresin is separated into two fractions: a volatile portion, or commercial gum turpentine, and a solid residue, or gum rosin. Because of their close association, but especially when fractionation is incomplete or faulty, gum rosin is apt to contain a varying, though generally small, percentage of ordinary turpentine. This explains partly why European observers have often found such a close correlation between sensitivity to adhesive tape and to turpentine, for the European products have contained, at least in some of the formulas used, a fair percentage of the latter substance. However, this is not the entire explanation, for the possibility of group reactions and multiple independent sensitivities must also be considered. In any event, the modern methods of preparing gum rosin seem to insure restriction of this contaminant to a negligible minimum. Nevertheless, this factor must always be considered in

evaluating the results of patch tests with gum rosin, especially the pale-colored specimens.

On the other hand, wood rosin is derived from pine stumps by solvent extraction followed by steam distillation. Certain grades of it are apt to contain products of phenol decomposition of wood and perhaps other irritants. This is especially true of the dark-colored wood rosins, which are, however, not likely to be used in adhesive tapes without subsequent purification. Wood rosin is generally free from turpentine, owing to more thorough fractionation. These differences, among others to be mentioned, between gum and wood rosin are probably significant in patch tests with these substances.

Neither wood nor gum rosins are compounds of constant composition, but within each class there are various grades, depending, for instance, on the source or on the method of preparation. Even specimens from the same tree may show chemical differences which may be important in the study of hypersensitivity to adhesive tapes.

The chief constituent of both rosins is abietic acid ($C_{19}H_{29}COOH$), a derivative of the phenanthrene group, which contains many remarkable substances of biologic importance.[6] Abietic acid probably does not exist as such in the original oleoresinous excretion of the pine tree but rather represents a more stable isomerized form of other primary acids. This change to the more stable form is induced by heat and by a combination of heat, water and acids, such as acetic acid. The degree of isomerization seems to vary with different samples. The crude abietic acid used in our study was obtained by heat treatment and acidification and in reality was a mixture of various acids, including abietic, pimaric and the probably heterogeneous pyroabietic acids,[7] together with oxidized residues which were said to be at least partially soluble in water. This complex substance was derived from gum rosin, which is apt to contain a greater mixture of such acids. Our crude product reacted to all the usual chemical tests for ordinary rosin, such as a positive Liebermann-Michel color reaction, a green copper abietate salt soluble in benzene and a white precipitate of abietic acid on acidification of an ammonium hydroxide fraction. On the other hand, the more purified abietic acid (known as Steel's acid) is a white crystalline substance which also turns yellow-brown on prolonged exposure to air, and this is usually obtained from wood rosin in an almost quantitative yield. It must be stressed that abietic acid and the other acids in rosin, whether primary

6. Fieser, L. F.: The Chemistry of Natural Products Related to Phenanthrene, New York, Reinhold Publishing Corporation, 1936.

7. Fleck, E. E., and Palkin, S.: On the Nature of Pyroabietic Acids, J. Am. Chem. Soc. **60**:921, 1938.

or secondary, are probably present in colloidal form, whereas the crystalline acids are obtained only after subsequent treatment. This point may have importance in patch tests with rosin and with pure crystalline abietic acid.

Abietic acid is soluble in most organic solvents and in oils but not in water. Some investigators have therefore postulated that when it acts as an irritant to the skin it does so by virtue of solubility in the oily secretion of the integument or through the medium of some organic solvent in the formula which keeps it in solution. We are not certain that this is the mechanism. In any event, no definite evidence has yet been furnished to indicate that this acid, either in crystalline or in colloidal form, is a frequent sensitizer of the skin. Our experiments indicate that this acid is not a primary irritant.

Abietic acid is an unsaturated substance with two conjugated double bonds, probably arranged between two adjacent rings of a phenathrene nucleus.[8] Because of its high degree of unsaturation, it is apt to be easily oxidized. It is said that in this way peroxides are formed and that these break down in the presence of moisture to form ultimately water-soluble fractions. If this is true, it may provide an explanation for the occasional discordant results of patch tests with rosin and abietic acid. In any event, this point needs to be investigated. Rosin and many of its derivatives are easily oxidized, with the result that there is depreciation in their adhesive qualities as well as catalytic changes (probably depolymerization) in any rubber that may have been incorporated in the adhesive tape. Manufacturers have therefore sought to counteract this by the use of small amounts of an antioxidant, as in transparent tape, or to seek other derivatives of rosin featured by absent or minimal oxidation, thus avoiding the necessity of adding an antioxidant.

Abietic acid contains one carboxyl group (monobasic). Chemists have long known of the relative difficulty in esterifying this group. The esterification of the three available hydroxyl groups in glycerin is generally accomplished by catalysis and heat, and the resultant compound is the so-called ester gum. The term ester gum is a generic one for all the triglycerides of rosin, whether the actual rosin was derived from the oleoresinous excretion of the trees or from pine stumps. These types of ester gums probably differ from one another in certain chemical respects, but precise knowledge on this point is still fragmentary. It is probable that all the fractions in the original rosin, including a small amount of unsaponifiable matter and other substances, are incorporated in the ester gum. It is also possible that in the manufacture of this

8. Fieser, L. F., and Campbell, W. P.: Concerning Dehydroabietic Acid and the Structure of Pine Resin Acids, J. Am. Chem. Soc. **60**:159, 1938.

compound a certain amount of decomposition may occur. These points may be important in studying hypersensitivity to ester gums, and that is the reason we made preliminary efforts to obtain fractions.

Ester gums are used in adhesive tapes for the following reasons: They furnish a relatively inexpensive compound retaining resinous properties; they are less likely to be easily oxidized; they raise the melting point, so that the adhesive is less apt to "run," and, finally, they are free from turpentine, which is alleged to have a deleterious effect on the aging of rubber. It may be mentioned that esterification with other higher alcohols, for example, pentaerythritol, seems to be coming into fashion. This fact is extremely important, for investigators may be deluded into believing that they are dealing with ordinary glycerinated ester gun. An instance of this confusion already exists in the literature, as will be mentioned in another part of the paper.

Ester gums are manufactured generally to have an acid number ranging between 3 and 8, that is to say, it requires from 3 to 8 mg. of potassium hydroxide to neutralize the free acid groups in 1 Gm. of substance. If one considers that the acid number of the original rosin varies between 154 and 168, this means a reduction in acidity of about 95 per cent, which is excellent neutralization in the case of organic acids. In actual practice, moreover, a more perfect neutralization, besides being difficult, has been found to be undesirable for certain commercial uses of the ester gum. If the actual irritant in the ester gum were free abietic acid, a more violent cutaneous reaction would be expected in patch tests with the original rosin, which contains far more abietic acid. This was, however, not the case in our patient. The ester gum (wood rosin) to which our patient was sensitive had an acid number of from 6 to 8, which indicated only a small amount of free acid. Our negative results with crude abietic acid appeared to indicate that neither this acid nor any of the companion acids in this product nor any of the extracts made from this compound was the actual cause of the dermatitis in our patient. It is realized that abietic acid as it exists in rosin may not be quite the same in its physicochemical properties as the pure crystalline substance or as even our crude abietic acid, and we must therefore suspend judgment on this point.

There are now being manufactured, also, many types of hydrogenated rosins and derivatives, the advantage of which lies in the relative or complete absence of oxidation. One of us (H. K.) has recently investigated a case of dermatitis due to adhesive tape, which was due, in part, to a commercial product containing a form of dehydrogenated abietic acid.

2. *Interpretation of the Patch Test.*—Our study seemed to shed light on a number of points of fundamental interest.

A. A patch test with the undiluted ester gum gave a more intense positive reaction than with the transparent tape itself. This was probably due to the application of the glycerinated ester gum in greater concentration than it existed in the commercial product. While, therefore, the original conditions in the adhesive plaster were not exactly reproduced, the data revealed by the patch tests were nevertheless conclusive in disclosing the actual irritant. The sensitivity shown by our patient to ester gum (wood rosin) recalls the recent report by Schwartz and his co-workers[9] on contact dermatitis due to a new resin finish used in articles of clothing, such as shorts, and stockings, the cause having been traced to the ester gum in the finish. However, the nature of the ester gum was not mentioned, nor was sufficient proof given to support the contention that abietic acid was the actual irritant. Sensitivity to an ester gum does not necessarily mean that abietic acid is the responsible agent. Furthermore, it was assumed by these investigators that the ester gum was the glycerinated type; but, on the basis of other investigations pursued by one of us (H. K.), it can now be stated that this substance is derived from esterification of wood rosin by an entirely different alcohol. The clinical manifestations of these various conditions which can be attributed, in large part at least, to ester gums of various types seem to depend on the degree of hypersensitivity of the patient, the method of manufacture of the product, the concentration of the offending substance or combinations of substances, the physicochemical properties of the compound, the extent of contact with the skin and the nature of the part of the integument to which the product is applied (for example, the presence of perspiration or numerous hair follicles). In addition, the skin of our patient showed a positive reaction to a patch test with gum rosin. Whether this was due to a group reaction to a similar substance, to some special contaminant or to an independent sensitivity we are unable to state definitely. Our investigations, however, warranted the conclusion that this positive reaction was not due to abietic acid, which is found in both gum rosin and the glycerinated ester gum; indeed, the reaction to gum rosin, which contains a high percentage of abietic acid, was less intense than that shown by the ester gum, which had only a small amount of free acid. As already stated, this comparison as to acid content may possibly not be valid in view of the possible differences in the physical state of abietic acid in these substances. The negative reaction to a patch test with gum turpentine indicated that this substance, which may occasionally

9. Schwartz, L., and others: An Outbreak of Dermatitis from New Resin Fabric Finishes, J. A. M. A. **115**:906 (Sept. 14) 1940.

contaminate gum rosin (grade WW), was not the cause of the positive reaction to gum rosin.

B. During the course of this study we used two other types of natural resins, namely, dammar resin and gum mastic. In addition, the rubber compounds are known to have small amounts of resinous matter. To all these substances the skin of the patient was not sensitive. This illustrates the important point that hypersensitivity to one type of natural resin is not necessarily accompanied by a like response to other varieties of resins.

C. The patch tests revealed, also, that there was no sensitivity to zinc oxide and orris, substances formerly reported as causes of dermatitis to adhesive tape. It is interesting that another compound, lead oleate, elicited a pronounced positive reaction. Since this was not present in the formula for the adhesive material and since there seems to be no obvious chemical relation between it and the ester gum, we must regard this positive reaction as coincidental and an example of multiple, independent sensitivity. Hence, the results of patch tests may be misleading unless one controls them with a precise knowledge of the actual ingredients in the commercial product.

D. The negative reactions to patch tests with the two nonimpregnated rubber compounds bears mention, for in this state these substances seem to be practically innocuous. However, some adhesive plasters contain smoke-cured rubber, and one of us (H. K.) has observed an instance in which this was one of the factors involved in the production of a dermatitis by adhesive tape. In drawing conclusions about the sensitizing power of rubber on the skin, the precise source and type ought to be mentioned.

E. A positive reaction to a patch test was obtained with a specimen of so-called patch tape adhesive, whereas another brand of plaster gave a negative reaction. We had no opportunity to study the chemical nature of these products for a number of reasons, the chief one being that we did not have the means to identify the ingredients in these tapes. Sensitivity to one adhesive is likely to be accompanied by sensitivity to another adhesive (in view of the possibility of group reactions to similar rosins), but this seems to be not necessarily so.

F. We were unable to demonstrate the presence of Prausnitz-Küstner (humoral) antibodies either to the transparent adhesive tape or to its ingredients. This conforms to the results obtained by practically all investigators who have been concerned with this phase of the subject. Perutz [10] apparently demonstrated local tissue antibodies (in the skin) to a European adhesive plaster, but, as his experiments were not clean-

10. Perutz, A.: Beiträge zur Klinik, Pathogenese und Therapie der Terpentin-Dermatitis, Arch. f. Dermat. u. Syph. **152**:617, 1926.

cut and the interpretation is subject to debate and as substantiation of his results has not yet been accomplished, we prefer to leave the matter *sub judice.*

SUMMARY AND CONCLUSIONS

We are recording what seems to be the first investigated case of true hypersensitivity to one of the ingredients in a transparent form of adhesive tape. The need for fractional patch tests in studying this type of dermatitis is stressed. The cause of the contact eczema in our case was an ester gum (glycerinated wood rosin); but while there is reason to believe that the reaction was due to the ester gum itself, we cannot exclude the possibility that the causative agent was some other fraction incorporated into this compound. Our investigations indicated, however, that abietic acid and the various components in the crude abietic acid used in this study were probably not the essential cause of the dermatitis. There was no correlation with hypersensitivity to turpentine. Prausnitz-Küstner (humoral) antibodies could not be demonstrated.

The cutaneous reactions to adhesive tapes were classified. The necessity for distinguishing simple mechanical irritation from true hypersensitivity was emphasized.

The differences between the various rosins and some of their derivatives were discussed.

Before a particular substance is designated as a poor or good sensitizer of the skin, its precise source must be mentioned together with other pertinent data. Thus, what holds for one gum rosin may not hold for another specimen of this compound, for wood rosin or for other derivatives of rosin; what applies to ordinary nonimpregnated rubber may not apply to the same rubber when it is smoke cured or vulcanized; what is true of fresh gum turpentine may not necessarily be true of oxidized gum turpentine, wood turpentine or rectified turpentine, for example. This point is of particular importance in investigating natural products of complex and variable composition.

Since formulas for adhesive tapes are occasionally changed and since there are difficulties in keeping such formulas constant even when the same ingredients are used, such points must be borne in mind in evaluating phenomena of the following types: (1) differences in the cutaneous reactions to apparently the same kind of adhesive tape applied at widely spaced intervals of time, (2) alleged examples of spontaneous desensitization and (3) other discordant results with "the ingredients of adhesive tape." While this may not explain all cases, owing to the possibility of additional factors, we believe that investigations along these lines should take cognizance of these variables.

A positive reaction to a patch test need not necessarily mean detection of the offending substance in the adhesive tape. Thus, our patient was

sensitive to gum rosin (probable group reaction) and also to lead oleate (multiple, independent sensitivity). The results of the patch test require careful correlation with the actual ingredients in the commercial product, based on a knowledge of the physicochemical attributes of the incorporated substances and their relation to one another. The cooperation of the manufacturer is often essential in such investigations.

Finally, it must be recognized that there are pitfalls in studying the reactions of skin to adhesive tapes, and these must be circumspectly evaluated before general conclusions are drawn.

Dr. E. V. Romaine gave advice on the chemical aspects of this study.

509 Madison Avenue, New York.

2209 Eutaw Place, Baltimore.

DERMATITIS OF THE EYELIDS DUE TO PHILODENDRON (SCANDENS CARDATUM) PLANTS

JOHN H. HARRIS, M.D.
NEW YORK

Dermatoses of the eyelids are frequently seen, especially in women. They are usually associated with involvement of other parts of the body, as in seborrheic dermatitis, psoriasis, neurodermatitis or lupus erythematosus. When a dermatitis involves only the eyelids it is usually considered to be of the contact type (due to an external irritant). The commonest irritants which cause such a dermatitis are mascara, eye shadow, perfume, various types of cosmetic creams and nail polish.

A review of the literature disclosed numerous allergens which may cause a dermatitis of the eyelids. Greenbaum [1] reported a case of a severe edematous vesicular dermatitis involving the eyelids from an eyelash dye. Rattner [2] described 4 cases in which dermatitis of the eyelids was due to hair tonic, wave lotion, fur collar and cold cream respectively. Hollander [3] observed a case in which hair tonic was the causative agent. In this case the hair tonic was used by the patient's husband, and contact with a head bolster on which both slept caused a dermatitis of the wife's eyes. Sutton [4] recorded a severe case of dermatitis of the eyelids due to "horn rim" spectacles; the rims were made of zylonite. Goldstein [5] treated a greenhouse employee who had several recurrences due to contact with chrysanthemum leaves. James [6] had a patient who used 1 per cent solution of procaine hydrochloride for conjunctivitis, which caused a dermatitis. Fox [7] reported a case in which the condition was due to the rubber on an eyelash curler. The eruption on the eyelids in this case recurred several times before the causative agent was deter-

1. Greenbaum, S. S.: Dermatoconjunctivitis Due to Lash-Lure, an Eyelash and Eyebrow Dye, J. A. M. A. **101**:363 (July 29) 1933.

2. Rattner, H.: Dermatitis of the Eyelids, J. A. M. A. **103**:180 (July 21) 1934.

3. Hollander, L.: Dermatitis of the Upper Eyelids Due to the Use of a "Hair Tonic," J. A. M. A. **97**:1384 (Nov. 7) 1931.

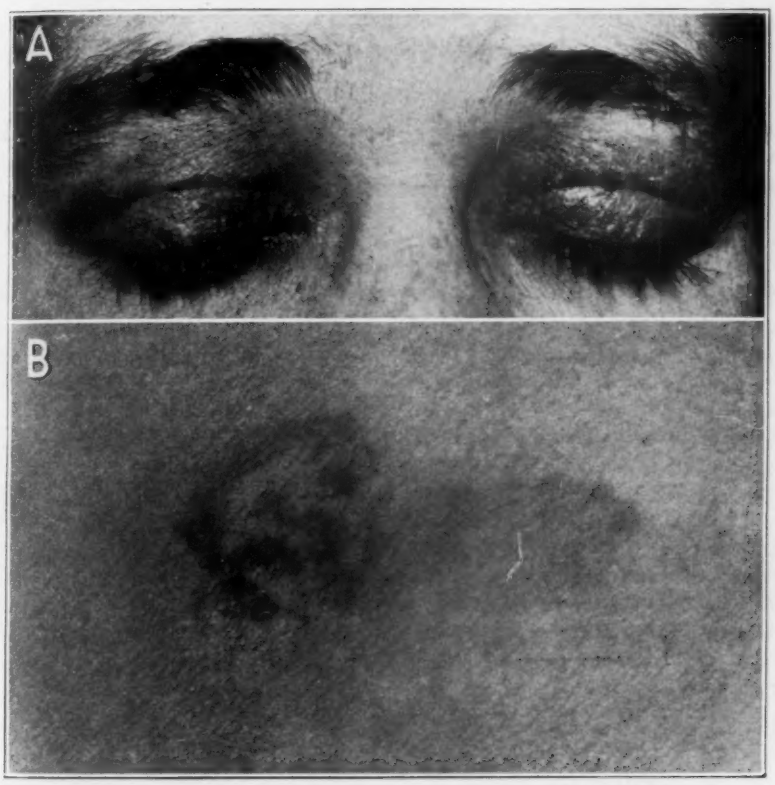
4. Sutton, I. C.: Acute Dermatitis from the Wearing of "Horn-Rim" Spectacles, J. A. M. A. **89**:1059 (Sept. 24) 1927.

5. Goldstein, M. B.: Dermatitis Venenata Due to Chrysanthemum Leaves, J. A. M. A. **96**:1680 (May 16) 1931.

6. James, B. M.: "Procaine Dermatitis," J. A. M. A. **97**:440 (Aug. 15) 1931.

7. Fox, E. C.: Dermatitis of the Eyelids Due to Rubber on Eyelash Curler, Arch. Dermat. & Syph. **28**:222 (Aug.) 1933.

mined. In the case reported by Cummer [8] the cause of the dermatitis proved to be 4 per cent ethylmorphine hydrochloride instilled in the eye. Hollander,[9] Ellis and Kirby-Smith,[10] Sulzberger [11] and others have reported cases of dermatitis of the eyelids due to nail polish. Feiler [12] reported a case in which this condition was due to perfume, lipstick and platinum nail polish.



A, dermatitis of the upper eyelids due to philodendron (Scandens cardatum) plants. *B*, site of patch test at the end of forty-eight hours, showing vesicular reaction.

8. Cummer, C. L.: Dermatitis of the Eyelids Caused by Dionin, Arch. Dermat. & Syph. **23**:68 (Jan.) 1931.

9. Hollander, L.: Nail Lacquer Dermatitis, J. A. M. A. **115**:1714 (Nov. 16) 1940.

10. Ellis, F. A., and Kirby-Smith, H.: Contact Dermatitis Due to Nail Polish, J. A. M. A. **116**:45 (Jan. 4) 1941.

11. Sulzberger, M. B.: Hypersensitivity to Nail Polish, Arch. Dermat. & Syph. **36**:460 (Aug.) 1937; Eczema Venenatum from Nail Polish, ibid. **42**:218 (July) 1940.

12. Feiler, H. B.: Eczema Venenatum Due to Lipstick and Nail Polish, Arch. Dermat. & Syph. **42**:224 (July) 1940.

REPORT OF A CASE

G. F., a white woman aged 23, had an eruption on the eyelids which developed in September 1940. From then until January 1941 the dermatitis had periodic recurrences and exacerbations. The latter appeared consistently in the early part of the week.

The patient was treated by two competent dermatologists each of whom made diagnosis of contact dermatitis. Patch tests were performed with cold cream, vanishing cream, soap, soap flakes, eye shadow, eyebrow pencil, lipstick, nail polish, dog hair and various articles of clothing. The reactions to all were negative. Treatment consisted of local applications and roentgen irradiation. The latter therapy was temporarily helpful.

Examination showed a pronounced swelling of the upper eyelids and to a much less degree of the lower lids. The skin was erythematous and somewhat stretched. There was a slight amount of scaling. In the longitudinal folds there were fissures, as shown in the accompanying illustration (*A*).

The Wassermann reaction of the blood was negative, and a complete blood count gave normal results.

Examination of the patient's home revealed that she had eight philodendron (Scandens cardatum) plants. A patch test was performed with a piece of the leaf of one of the plants. After twenty-four hours the patch was removed, with no evidence of any reaction. At the end of forty-eight hours the site of the patch test presented a definite erythematovesicular reaction, as shown in the illustration (*B*).

On being questioned, the patient stated that in March 1940 she had bought one plant and in September she had purchased seven more (the eruption appeared the latter part of September). Further questioning showed that the patient washed and watered these plants every week end, which probably accounted for the exacerbations of the dermatitis in the early part of the week. The plants were removed from her home immediately, and within a few days the edema began to subside. She was not entirely well, however, until two months later. The only treatment was application of a bland ointment (boric acid ointment). It was suggested that the patient attempt another exposure to the plants in order to ascertain if she was still sensitive, but she refused to cooperate.

SUMMARY

A case of dermatitis of the eyelids due to a house plant, Philodendron (Scandens cardatum), is reported with a view to including this in the list of cutaneous irritants. A review of the literature failed to reveal any report of a localized dermatitis of the eyelids or other parts of the body caused by this plant.

30 East Seventy-Second Street.

CUTANEOUS MANIFESTATIONS OF INTERNAL MALIGNANT TUMORS

S. WILLIAM BECKER, M.D.

DAVID KAHN, M.D.

AND

STEPHEN ROTHMAN, M.D.

CHICAGO

A survey of the cutaneous disturbances accompanying malignant tumors of internal organs was presented first in 1925.[1] The changes observed up to that time were classified in four groups: (*a*) pruritus and a prurigo-like syndrome with hyperpigmentation, (*b*) toxic-bullous eruptions, (*c*) metastatic cutaneous lesions and inflammatory lesions preceding metastases and (*d*) acanthosis nigricans and similar proliferative changes.

Simple pruritus and prurigo-like eruptions with hyperpigmentation have been observed frequently in cases of internal malignant tumor. The disappearance of the cutaneous manifestations after removal of the tumor and their recurrence with the reappearance of the tumor, as observed in single cases, seemed to prove the causal connection between tumor and eruption.

The toxic, for the most part bullous, eruptions described were manifold in their clinical aspect and could not be recorded as belonging to certain definite types. They simulated erythema multiforme, dermatitis herpetiformis, acute lupus erythematosus and exfoliative dermatitis. Herpes zoster occurred in some cases. In 1 case the dermatitis-herpetiformis-like eruption appeared after roentgen treatment of sarcoma of the thyroid.

Some of the inflammatory eruptions were interpreted as being not of toxic but of metastatic origin. It was assumed that single tumor cells transported to the skin by the blood stream are decomposed in the skin with accompanying local inflammation. Evidence for such a process was seen in cases in which metastases developed from primarily itching papular inflammatory lesions.

From the Section of Dermatology, University of Chicago, The School of Medicine.

1. Rothman, S.: Ueber Hauterscheinungen bei bösartigen Geschwülsten innerer Organe, Arch. f. Dermat. u. Syph. **149**:99-123, 1925.

Transitions from noncharacteristic itching eruptions to acanthosis-nigricans-like changes have been described, and it was assumed that a growth-promoting principle is contained in the toxic products of the tumor which causes such eruptions, furthering the tissue overgrowth seen in acanthosis nigricans (dystrophia papillaris et pigmentosa).

Cutaneous changes due to internal malignant tumors seem to be rare, but they may occur as a consequence of any kind of malignant tumor of any localization, mostly as late manifestations and usually after considerable necrosis of the tumor cells has occurred. Pruritus and hyperpigmentation occur more frequently with carcinomas of the gastro-intestinal tract than with growths in other localizations.

A survey of the literature since 1925 reveals the publication of but 8 cases demonstrating a relation between dermatoses and malignant tumors. In a case of round cell sarcoma of the thyroid gland, Ramel [2] observed a toxic erythematopapulo-urticarial and hemorrhagic eruption which developed immediately after the appearance of a cutaneous metastatic lesion and lasted for five days only but reappeared shortly before death. In a case of round cell sarcoma of the thyroid gland [2] a dermatitis-herpetiformis-like eruption was noticed.

Itching as a premonitory symptom of cancer of the breast was observed by Verhave.[3] The pruritus started several months before the tumor was noticed.

S. Kitamura [4] reported a case of branchiogenic cancer in which a pigmented, scaling, slightly pruritic eruption on the body and erysipelas-like changes on the face appeared four months before the tumor in the left submaxillary region became clinically evident. The edema of the face disappeared and reappeared four times during the period of observation. The hyperpigmentation of the neck was perifollicular.

The patient of Usland,[5] suffering from scirrhous carcinoma of the stomach, had generalized itching for nine months and rapidly progressing dermatitis herpetiformis for five months. In addition to the dermatitis herpetiformis, acanthosis-nigricans-like changes were present in the axillas and on the neck with pronounced pigmentation and papillary hypertrophy. After resection of the stomach the eruption disappeared within one month.

---

2. Ramel, E.: Contribution à l'étude des dermatoses toxiques survenant au cours de tumeurs malignes, Schweiz. med. Wchnschr. **56**:790-792, 1926.

3. Verhave, J. H.: Itching as Precursor Phenomenon of Mammary Cancer, Nederl. tijdschr. v. geneesk. **70**:1082-1083, 1926.

4. Kitamura, S.: Branchiogener Krebs und eigentümliche universale Hautexantheme, Jap. J. Dermat. & Urol. **27**:702-708, 1927.

5. Usland, O.: Skin Eruption as Premonitory Symptom in Gastric Cancer, Med. rev., Bergen **44**:13-23, 1927; abstracted, Zentralbl. f. Haut- u. Geschlechtskr. **24**:369, 1927.

Beron [6] reported a case of "pellagroid" eruption in a patient with cancer of the stomach. The eruption started with erythema and scaling on the neck, later involving the face, the dorsa of the hands and the upper parts of the chest and back. Pigmentation and hyperkeratosis developed, particularly evident around the follicles. The eruption did not resemble acanthosis nigricans.

Wartenberg [7] reported 2 cases of tumor of the brain with increased intracranial pressure. In both cases there was intense pruritus of the nostrils, a remarkable localization in view of the results of animal experiments in which the nostrils are sites of predilection for itching of central origin.[8]

These case reports published since 1925 confirm earlier observations [1] concerning the occurrence of itching, hyperpigmentation, toxic eruptions and hyperplasia of tissue (acanthosis nigricans) in connection with the development of internal neoplasms.

REPORT OF CASES

CASE 1.—Mrs. C. F., a Polish housewife aged 45, was admitted to the University of Chicago clinics on Jan. 2, 1940 with a widespread symmetric eruption which had started eight months previously. She had lost 17 pounds (7.7 Kg.) during this period and had noticed increasing weakness for several months. In May 1939 a pruritic erythematovesicular eruption with exudation appeared on the dorsa of the feet. Four exposures to roentgen rays and the application of numerous ointments gave only temporary relief. After one or two months, red scaly "spots which later turned brown" extended up the leg toward the groin. One month prior to admittance, similar lesions appeared on the sides of the chest and later in the anal crease, in the groins and about the vulva. A finely vesicular patchy eruption then appeared on the dorsa of the hands and fingers. All the lesions were so pruritic that at times they interfered with sleep.

Other complaints included soreness of the tip of the tongue, tingling of the feet, nervousness and irritability, constipation and occasional "burning" in the left side of the chest. There were nocturia, urinary frequency and burning after urination, the latter having appeared since the vulva had become involved. The menses had stopped eight months previously. There was no family history of diabetes or cancer. The patient had previously had a "milk leg."

Examination revealed symmetric diffuse erythema on the dorsa of the hands, fingers and feet with tiny vesicles and serous crusting. The dorsum of the right foot exhibited a punched-out ulcer, 0.75 to 1 cm. in diameter, with a fairly clean granulating base and an elevated firm rolled border. The finger nails and toe nails were distorted, with pitting longitudinal striae and deposits of yellowish white grumous material beneath the nails.

---

6. Beron, B.: Pellagroide Hautveränderungen und Magencarcinom, Zentralbl. f. Haut- u. Geschlechtskr. **35**:457, 1931.

7. Wartenberg, R.: Pruritus nasi bei Hirntumoren, Klin. Wchnschr. **11**:461-462, 1932.

8. Rothman, S.: Physiology of Itching, Physiol. Rev. **21**:357-381, 1941.

Macular erythema with few large scales was present in widespread patchy distribution on the thighs and legs. Annular erythema with central clearing and small sanguineous crusts occurred below each patella. The groins, gluteal folds and vulva exhibited diffuse erythema, edema and serous crusting. Numerous scattered patches consisting of papules and macular lesions with some brownish

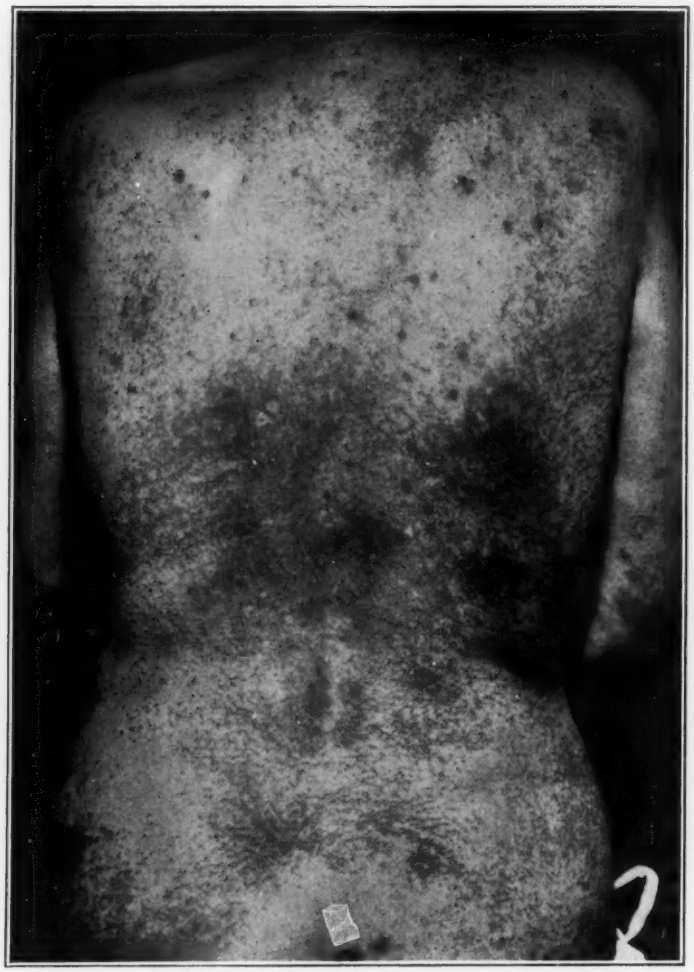


Fig. 1 (case 1).—Widespread purpuric inflammatory eruption.

crusts were present on the trunk. There was perioral perlèche. The tip and the lateral borders of the tongue were red, smooth and tender.

The blood pressure was 116 systolic and 74 diastolic; the temperature, 99 F.; the pulse rate, 108, and the respiratory rate, 20. The results of the remainder of the physical examination were irrelevant.

Monilia was demonstrated in the nail scrapings and later cultured in material from the lesions of perlèche and from those in the groin and on the tongue.

Repeated cultures of material from the other cutaneous lesions and from those developing subsequently showed no organisms. The routine intracutaneous dermotrichophytin (Bischoff) test gave a strongly positive reaction.

During six weeks in the hospital, the dermatosis at first became more widespread with extension of the pruritic erythematous macular lesions over the trunk anteriorly and posteriorly. Many of these lesions then became purpuric (fig. 1), and later large confluent patches showing superficial necrosis resulted (fig. 2). The necrotized epidermis formed a thin dark crust which was cast off in a few days, and healing occurred with hyperpigmentation. These necrotic lesions resembled a superficial chemical burn in their appearance and course. Meanwhile an erythematous papulovesicular eruption appeared on the cheeks and across the

Fig. 2 (case 1).—Confluent patches showing superficial necrosis. Exfoliation of this necrotized epidermis had occurred on the inner aspect of the left thigh.

bridge of the nose. As this area cleared, a similar eruption gradually spread upward to involve the eyelids and forehead (fig. 3). Repeated cultures of material from these lesions were negative for fungi. Gradual involution of all the cutaneous changes then occurred.

A biopsy specimen from an area of superficial necrosis on the abdomen (fig. 4) showed the following picture: The pathologic changes were confined to the superficial portion of the section. The stratum corneum was thick, parakeratotic and acidophilic throughout. In one place it had been loosened by a collection of serum. The stratum mucosum was of about normal thickness and showed an increased number of mitotic figures in the basal portion. The papillae were edematous; the capillaries were dilated, and the endothelial cells were swollen.

The vessels of the superficial cutis also showed endothelial swelling, perivascular edema and moderate perivascular infiltrate with round cells. The deeper portions of the section were essentially normal. A diagnosis of subacute dermatitis was made.

The following features were observed in a biopsy specimen from a crusted lesion on the chest (fig. 5): The stratum corneum was greatly thickened, parakeratotic and separated into several layers by collections of serum and remnants of what appeared to have been mononucleated cells. The stratum corneum had been separated from the stratum granulosum in some places by serum. The

Fig. 3 (case 1).—Erythematous papulovesicular and crusted lesions of the eyelids and forehead.

stratum granulosum was irregularly thickened and thinned and in some places showed liquefaction necrosis with practical disappearance. There was intercellular and intracellular edema, chiefly the former. There was an abnormally large number of mitotic figures in the basal portion. The papillae were edematous. The papillary and subpapillary layers of the cutis were involved in polymorphonuclear leukocytic infiltration. The blood vessels showed endothelial swelling and a perivascular round cell infiltrate. A diagnosis of acute and subacute exudative dermatitis was made.

Laboratory examination revealed a normochromic anemia (3,520,000 red blood cells per cubic millimeter and 11 Gm. of hemoglobin per hundred cubic centimeters) and 7,400 leukocytes, with 60 per cent polymorphonuclear neutrophilic leukocytes and 3 per cent eosinophilic leukocytes. Urinalyses showed occasional 1 plus reduction on a high vitamin diet without insulin. The fasting blood sugar value on two occasions was 83 mg. and 129 mg. per hundred cubic centimeters. A dextrose tolerance test gave the following results: fasting blood sugar, 83 mg. per hundred cubic centimeters; ½ hour, 141 mg. (with a 3 plus reaction for sugar in the urine); 1 hour, 210 mg.; 2 hours, 162 mg. (with a 4 plus reaction for sugar in the urine), and 3 hours, 125 mg. (with a trace of urinary sugar).

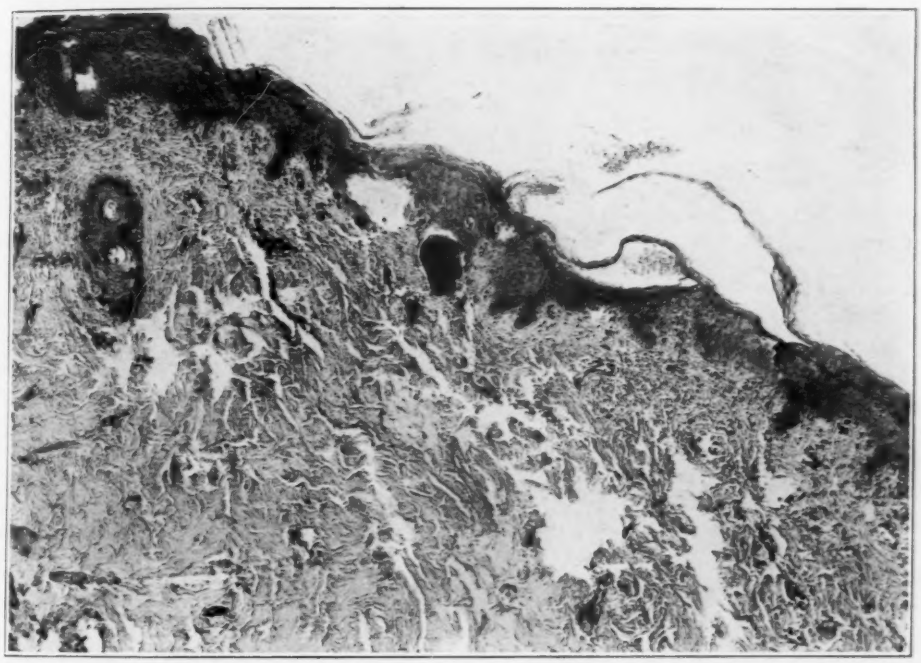


Fig. 4 (case 1).—Section from an area of superficial necrosis on the abdomen, shown in figure 2 (low power magnification; hemalum-erythrosin-saffron stain).

Local treatment consisted of local applications, roentgen irradiation and ultraviolet irradiation; systemic medication consisted of iodides, ferrous sulfate and a high vitamin diet, reenforced by vitamin B complex and ascorbic acid. The anemia persisted (hemoglobin, 10 to 11 Gm.) despite iron therapy and two transfusions. The white blood cell count was 6,000 to 8,400 at first and later 11,500, with 60 to 70 per cent polymorphonuclear neutrophils and 0 to 3 per cent eosinophils.

The first determination of plasma proteins revealed a low value (5.68 Gm. per hundred cubic centimeters with 3.12 Gm. of albumin); later the value was normal. The cholesterol content of the blood was 136 mg. per hundred cubic centimeters. The fasting ascorbic acid content of the plasma was extremely low (0.3 mg. per hundred cubic centimeters). The Kolmer and Kahn reactions of the blood serum were

negative. Cultures of the blood showed no organisms. Examination of the stool showed no abnormalities. There was no free acid in the gastric juice after administration of histamine. Additional hematologic studies, a urea clearance test and additional chemical tests of the blood added little information. Two roentgenograms of the chest (the last on Feb. 10, 1940) were normal.

At first the temperature was normal or subnormal. After a transfusion on January 17 it rose to 102 F., and from this time on there was usually fever, at first with a temperature of 99 to 100 F. and later 101 to 103 F. daily. Late in January cystitis was diagnosed and Bacillus proteus was cultured from the urine. On February 3 purulent otitis developed on the right side, and hemolytic streptococci and Staphylococcus aureus were cultured. Throughout the course of hos-

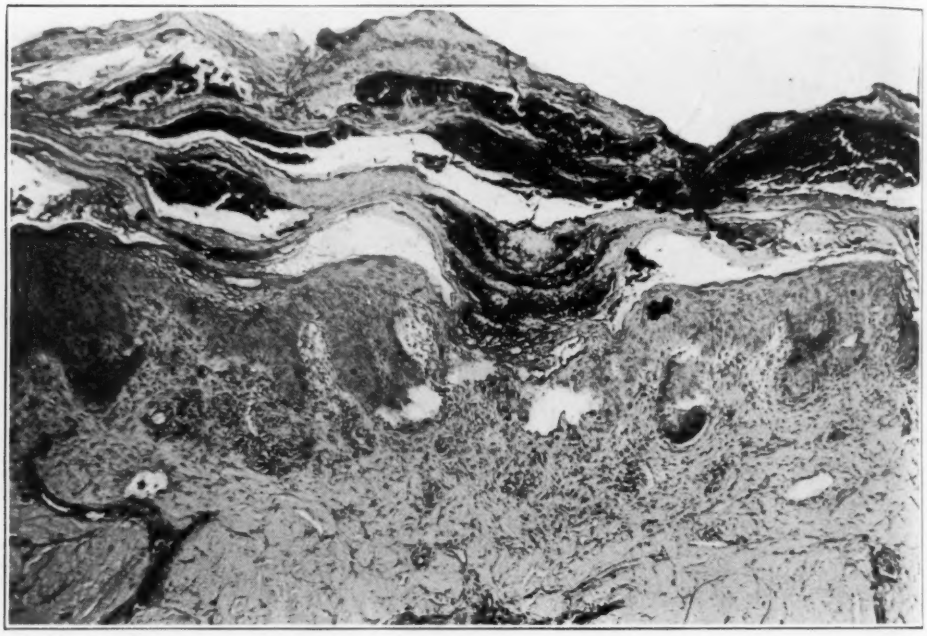


Fig. 5 (case 1).—Section from a crusted lesion on the chest (low power magnification; hemalum-erythrosin-saffron stain).

pitalization much sedation was required. About February 8 toxic psychosis developed.

Although the skin cleared considerably, no general improvement was evident. However, the termination was rather sudden. During the hospital stay there developed an unusual tendency of the patient's blood to clot rapidly (clotting time on admission, two minutes and thirty seconds, and on February 7, thirty-four seconds). Her left leg became swollen and dusky overnight, and the arteries, pulseless. Then the arteries of the right leg became pulseless. Acute thrombosis of the left iliac vein was diagnosed. Death ensued several hours later, on February 14, the picture being that of circulatory collapse and respiratory failure.

Postmortem examination revealed a neoplasm replacing the body and tail of the pancreas, with invasion of the adjacent lymph nodes and the left adrenal gland.

Microscopic study suggested the islet cell type of pancreatic carcinoma. Subacute cystitis and chronic atrophic gastritis were pronounced. There were recent thromboses of the inferior vena cava and left iliac and femoral veins and a membranous remnant at the mouth of the left common iliac vein. At the time of autopsy it was felt that the sudden death was not explained by the anatomic findings. One explanation attributed death to a "shock phenomenon." Edema developed with unusual rapidity, because of the accelerated clotting and the mechanical barrier where the left iliac vein joined the cava (old thrombus). The quick pouring out of fluid and protein into the leg depleted the blood stream as effectively as a sudden hemorrhage, with resultant shock and death.

CASE 2.—Mrs. M. G., an American housewife aged 62, entered the University of Chicago clinic on Aug. 27, 1940 with a diffuse erythematous scaly eruption on the scalp, face, neck, chest and arms. In June 1940 diffuse erythema with pruritus had appeared across the front of the neck. Temporary disappearance had resulted when wet compresses and an antipruritic lotion were employed. Similar lesions had then spread up the sides of the face, and soon the entire face had become diffusely red with moderate scaling and thickening of the skin. The scalp had become scaly with gradual loss of all the hair, followed by slow regrowth. The erythema and scaling had then spread downward over the chest to about the midsternal level. The patient also had large atrophic patches on the chest; the date of onset of these lesions was unknown. A few crusted lesions were present on the chest and in the cubital fossae, the latter starting as red firm "lumpy" lesions. The pruritus, which was of moderate intensity at first, soon became negligible. Swelling and redness of the eyelids and lacrimation were pronounced, but the lids were never swollen shut. There was no history of ingestion of drugs.

Despite a moderately good appetite and balanced diet, the patient had lost 35 pounds (16 Kg.) during the three months prior to admission and had noted progressive weakness. Dyspnea on exertion had been present for several years. The menses had ceased twelve years previously. Her husband had died of cancer.

Examination of the skin revealed vivid erythema of the entire face with moderate thickening and large lamellated scales, the latter particularly about the forehead and sides of the face. The scalp was erythematous and completely covered with thick white scales. A thin even growth of soft white hair was present. The eyelids were erythematous, thickened and edematous.

Diffuse erythema with large scales covered the neck and the upper part of the chest. Scattered crusted lesions and depigmented patches of atrophic scarring of varying sizes appeared on the chest. In the cubital fossae there were symmetrically placed extensive shallow ulcers with crusting and purulent exudate. A small group of erythematous macules was present in the right lower quadrant.

The tongue was smooth and red over the midportion, but there was no tenderness.

The blood pressure was 146 systolic and 78 diastolic (left leg). Slight cardiac enlargement to the left and a basal systolic murmur were present. The liver was 3 cm. below the costal margin. The abdomen was obese and somewhat distended. No masses were palpated.

On admission, the temperature was 100 F.; the pulse rate, 90, and the respiratory rate, 18. The urine showed no abnormalities. Blood counts revealed leukocytosis (12,000 to 18,000 leukocytes, with 85 to 98 per cent polymorphonuclear neutrophilic leukocytes) and moderate normocytic, slightly hypochromic anemia (59 to 70 per cent hemoglobin and 3,600,000 to 4,500,000 erythrocytes). The Kolmer and Kahn reactions of the blood serum were negative. Culture of the blood

showed no organisms. Roentgenograms of the chest were not entirely satisfactory because of the patient's weakness, but cardiac enlargement and an elevated diaphragm were definite. The patient remained too weak for us to attempt roentgen ray examination of the gastrointestinal tract.

During three weeks' hospitalization several new superficial oval ulcers developed on the chest and forearms and later on the shoulders and arms. Relatively rapid healing of these lesions occurred with atrophic scarring. After two weeks the erythema of the face diminished greatly, with that of the chest decreasing to a less extent.

Biopsy of an erythematous lesion on the face showed chronic inflammation. There was a thin parakeratotic stratum corneum. The stratum granulosum was absent in most of the section. The stratum mucosum was of normal thickness and showed moderate intercellular edema. The papillary portion of the cutis was edematous and contained some serum. In the deeper portion of the section there was a moderate amount of degenerating collagen. The sebaceous glands were adenomatous.

A biopsy specimen from an atrophic area on the chest showed only simple atrophy of the epidermis.

The patient received digitalis, intensive vitamin B complex therapy, antianemic medication and local treatment of the cutaneous lesions. She had an irregular rapidly changing temperature (ranging from subnormal to 104 F.), which subsided several days before death. Tachycardia persisted. An electrocardiogram showed left axis deviation. She lost her appetite, and urinary retention developed with overflow incontinence and cystitis. Bacillus coli was cultured from the urine, and sulfanilamide therapy was instituted. The total protein content of the plasma was low (5.56 Gm. per hundred cubic centimeters) with increased globulin (2.92 Gm.). Determinations of fasting blood sugar and serum calcium and phosphorus and urea clearance tests added little information. Repeated examination of the stools revealed a persistent 4 plus benzidine reaction while the diet was meat free.

Dyspnea increased and moderate acidosis developed (serum carbon dioxide, 18.6 millimols per liter; serum chlorides, 97.2 millimols per liter; serum p_H, 7.29, and nonprotein nitrogen, 38 mg. per hundred cubic centimeters). The patient continued to become weaker and died on Sept. 18, 1940.

Significant postmortem observations included a carcinoma of the cardiac end of the stomach with extensive necrosis (with compression of the spleen), multiple benign gastric ulcers, empyema of the gallbladder and hemorrhagic cystitis.

COMMENT

The interpretation of case 1 is extremely difficult because of the complexity of the pathogenic factors (pancreatic neoplasm, metabolic changes, signs of deficiency disease and monilia infection) and because of the multiplicity of the cutaneous changes. These lesions do not correspond to those observed in other cases in which there were eruptions due to internal neoplasms. Although the two cardinal symptoms of such eruptions—intense pruritus and accentuated hyperpigmentation—were also present in our patient, the morphologic aspect and the course of the eruption seemed to be unique, not only in connection with internal neoplasms but also in general. Especially, the diffuse progressive

epidermal necrosis with consecutive hyperpigmentation similar to a chemical burn seemed to be an entirely new manifestation, which up to the present time has not been described as occurring in moniliasis or in any dermatosis of internal origin.

Cutaneous changes due to pancreatic neoplasm have not been described as yet, and the peculiarity of the eruption may be due to the site of the tumor in the pancreas. It is probable that the disturbance in carbohydrate metabolism (mild borderline diabetes) was due to the neoplasm and that the monilia infection might have been a consequence of the metabolic disturbance. A further consequence of pancreatic insufficiency might have been deficiency symptoms. The following observations are from the autopsy report by Dr. Humphreys, of the Department of Pathology:

> There is no direct evidence (diarrhea or fatty stools) pointing to an exocrine defect. However, it is not impossible that there was some digestive-absorptive defect. The character of lesions of the lip and tongue and of some of those on the skin, the atrophic gastritis and the slight metaplasia in the tongue, esophagus and pancreatic ducts all suggest deficiency disease. Also, the improvement in the cutaneous lesions while the patient was on the vitamin-reenforced diet should be noted. There is no doubt that moniliasis (superficial) was responsible for some of the lesions; however, it was probably secondary—superimposed on mild diabetes and/or a deficiency disease.

Thus, a comment on our first case seems to be impossible at present, and we restrict ourselves to the recording of it. Only the observation of other cases of pancreatic tumor will decide whether the cutaneous changes in our case were primarily due to the neoplasm.

In case 2, the patient suffering from an internal carcinoma had an erythematosquamous eruption which responded favorably to external therapy. In this repect this case seems to be similar to that of Gougerot and Rupp,[9] in which an exfoliative dermatitis accompanying an internal carcinoma was cured by topical treatment. However, in our case ulcerations also developed within the area of the exfoliative dermatitis. Development of cutaneous ulcers in a patient with internal neoplasms was observed by Heuss.[10] In this case the ulcers developed from pruritic papules and later were identified as tiny carcinomatous metastases. In our case, however, the ulcers appeared in the areas of scaling erythema without previous formation of papules, and no carcinoma could be found histologically. Nevertheless, the unmotivated appearance of these ulcers and their rapid healing suggests that they may have been of metastatic origin.

---

9. Gougerot, H., and Rupp: Dermatose érythémato-squameuse avec hyperkératose palmo-plantaire, porectasies digitales et cancer de la langue latent. Contribution à l'étude des dermatoses monitrices de cancer, Paris méd. **12**:234-237, 1922.

10. Heuss, cited by Rothman.[1]

The survey of the literature strongly suggests that carcinoma cells of metastatic origin might be destroyed in the skin by cutaneous defense mechanisms and that the clinical manifestations of this defense reaction might be either necrosis or inflammation. Definite conclusions are not possible because of the extreme rarity of such observations. In the future it would seem to be desirable to observe patients suffering from malignant tumors more closely from the dermatologic point of view, especially in the late stages of the disease, since the cutaneous manifestations always appear to be late signs. The formation of cutaneous ulcers seems to be an even later manifestation, usually occurring shortly before death.

SUMMARY

1. A brief survey of cutaneous manifestations accompanying malignant tumors of internal origin is presented.

2. Cutaneous manifestations in 2 patients with internal neoplasms are described, and the possible relation between tumor and cutaneous processes is discussed.

950 East Fifty-Ninth Street.

LICHEN PLANOPILARIS

LICHEN PLANUS ET ACUMINATUS ATROPHICANS (FELDMAN) AND LICHEN SPINULOSUS AND FOLLICULITIS DECALVANS (LITTLE)

WILBERT SACHS, M.D.
JERSEY CITY, N. J.
AND
GERARD De OREO, M.D.
CLEVELAND

In this communication we report 6 cases of lichen planopilaris. We feel that the disease called lichen spinulosus and folliculitis decalvans (Little) and lichen planus et acuminatus atrophicans (Feldman) is one and the same as that recognized and fully described by Pringle (lichen planopilaris). We should like to emphasize that follicular lichen planus (lichen planopilaris) when it affects the scalp may simulate the end stage of pseudopelade (Brocq).

In 1895 Pringle [1] reported that follicular lesions were to be seen in lichen planus, and he called this condition lichen planopilaris (follicular type of lichen planus). He observed a patient with "a patch of lichen planus, the size of half a crown, on the right arm . . . and a band of lichen pilaris on either side of the neck." Two years later he [2] reported a similar case. A third case was that of a 69 year old man who had, in addition to follicular lesions in the supraclavicular areas, similar patches on the scalp. While he was under observation lichen planus appeared on the inner aspect of each knee, justifying the original diagnosis.

Even a cursory examination of the English literature of that period has disclosed several additional cases of similar nature. In the case described by Fox [3] there were ". . . acuminate papules with protruding horny spines seated at the pilo-sebaceous follicles. This eruption was sparse except on the legs, where every follicle seems to be affected. On the legs were a few typical plane papules, violaceous, flat-topped, polygonal lesions with a central orifice. . . . The buccal mucous mem-

From the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, New York.

1. Pringle, cited by Adamson, H. G.: The Relation of Lichen Spinulosus with Other Affections Having Follicular Spines or Plugs, Brit. J. Dermat. **17**:77, 1905.

2. Pringle, J. J.: Lichen Plano-Pilaris: Report of a Case, Brit. J. Dermat. **9**:74, 1897.

3. Fox, C.: Acuminate Form of Wilson's Lichen, Brit. J. Dermat. **14**:91, 1902.

brane was thickly studded with pearly papules." In that year Morris [4] reported a similar case. In 1904 Fox [5] described his second case. Other reports of the same process were later made by Ormerod,[6] by Saville [7] and again by Pringle.[8] Fordyce [9] cited 1 of Pringle's cases in a paper he wrote on lichen planus wherein he described follicular lesions.

In 1915 Little [10] described a patient with follicular lesions on the scalp and body. He stated the belief that the involvement of hairy skin and of glabrous skin constituted two distinct entities and therefore made the diagnosis of lichen spinulosus and folliculitis decalvans. Soon after, other cases were reported by Dore [11] and Beatty.[12] They accepted Little's original diagnosis. In 1919 Feldman [13] presented a case at a meeting of the Manhattan Dermatologic Society, and in 1922 he reported this as one of "lichen planus et acuminatus atrophicans." In 1921 Little [14] presented another case but now realized that his 2 previous cases (as well as those of Dore and Beatty) were instances not of lichen spinulosus and folliculitis decalvans but of a variety of lichen planus. In addition to the aforementioned reports, others have been described by Ormsby,[15] McCafferty,[16] Traub and Tolmach,[17] Feldman,[18] Corsi,[19] Fruchtbaum [20] and Ellis and Kirby-Smith.[21]

---

4. Morris, M.: Lichen Planus: Report of a Case, Brit. J. Dermat. **14**:94, 1902.

5. Fox, C.: Lichen Planus of Wilson with Acuminate and Plane Lesions, Brit. J. Dermat. **16**:340, 1904.

6. Ormerod, J.: Lichen Planus with Acuminate Papules: Report of a Case, Brit. J. Dermat. **14**:268, 1902.

7. Saville, T.: Lichen Plano-Pilaris: Report of a Case, Brit. J. Dermat. **20**: 234, 1908.

8. Pringle, J. J.: Lichen Plano-Pilaris: Report of a Case, Brit. J. Dermat. **17**:264, 1905.

9. Fordyce, J. A.: The Lichen Group of Skin Diseases: A Histological Study, J. Cutan. Dis. **28**:57, 1910.

10. Little, E. G.: Lichen Spinulosus et Folliculitis Decalvans: Report of a Case, Brit. J. Dermat. **27**:183, 1915.

11. Dore, S. E.: Lichen Spinulosus et Folliculitis Decalvans: Report of a Case, Brit. J. Dermat. **27**:295, 1915.

12. Beatty, W.: Folliculitis Decalvans et Lichen Spinulosus: Report of a Case, Brit. J. Dermat. **27**:331, 1915.

13. Feldman, S.: Lichen Planus et Acuminatus Atrophicans, Arch. Dermat. & Syph. **5**:102 (Jan.) 1922.

14. Little, E. G.: Lichen Spinulosus et Folliculitis Decalvans: Report of a Case, Proc. Roy. Soc. Med. **14**:67, 1921.

15. Ormsby, O. S.: Folliculitis Decalvans and Lichen Spinulosus, Arch. Dermat. & Syph. **1**:471 (April) 1920.

16. McCafferty, L. M.: Folliculitis Decalvans et Atrophicans (Little), Arch. Dermat. & Syph. **18**:514 (Oct.) 1928.

17. Traub, E. F., and Tolmach, J. A.: Folliculitis Decalvans and Lichen Spinulosus: Report of a Case, Arch. Dermat. & Syph. **24**:101 (July) 1931.

(Footnotes continued on next page)

REPORT OF CASES

CASE 1.—W. B.,[22] a salesman aged 47, born in Hungary, was first seen at the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital on Feb. 25, 1936, with an eruption of five months' duration involving the scalp, trunk and extremities.

Scattered over the back, chest, arms and thighs were numerous discrete follicular papules with keratotic plugs. There were occasional glossy, flat-topped papules with central depressions, more numerous on the forearms. On the lower portion of the abdomen (involving the pubic hair zone) and to a lesser degree on the upper middle part of the back was a cluster of atrophic macules. There were flat-topped, shiny, violaceous papules in both these areas. The oral mucous membrane was normal.

There was an irregular elongated scar on the vertex of the scalp, resulting from an injury which had occurred in childhood. In addition, there were several well defined patches of smooth alopecia in which the skin appeared atrophic. There had never been any pustules associated with the process. During the last five months other areas of alopecia developed in the frontal, parietal and temporal regions. The hair of the axillas was normal.

Tissue was excised for histologic examination from the margin of the patch of alopecia on the scalp and from a flat-topped shiny lesion on the trunk. The observations were the same in the two specimens; a disklike acanthosis; disturbance, disorganization and washing out of the cells of the basal layer, and increased granular and horny layer. Beneath the epidermis was a bandlike cellular infiltration composed chiefly of small round cells. Between the infiltration and the edematous lower border of the epidermis was a space (Max Joseph's lacuna). The section from the scalp showed a similar picture except that it was about the follicles. The base of the follicle was flattened and the hair papilla obliterated (figs. 1 and 2). Elastic tissue stains showed displacement and some fragmentation of the elastic fibers in the area of the cellular reaction.

CASE 2.—F. M.,[23] a man aged 53, came to the Skin and Cancer Unit in September 1938 with an eruption of the scalp, face and neck of one year's duration.

The entire scalp was uniformly involved by a violaceous papular eruption. Scattered over the scalp were areas of alopecia and atrophy varying from 0.5 to

---

18. Feldman, S.: Lichen Planus et Acuminatus Atrophicans: Folliculitis Decalvans et Lichen Spinulosus of Little, Arch. Dermat. & Syph. **34**:378 (Sept.) 1936.

19. Corsi, H.: Lichen Planus Associated with Atrophy of Nail Matrix and Hair Follicles on Scalp, Proc. Roy. Soc. Med. **30**:198, 1937.

20. Fruchtbaum, L. M.: Lichen Planopilaris (Pringle), Arch. Dermat. & Syph. **42**:156 (July) 1940.

21. Ellis, F. A., and Kirby-Smith, H.: Lichen Planus et Acuminatus Atrophicans (Feldman): Folliculitis Decalvans et Lichen Spinulosus (Little), Arch. Dermat. & Syph. **43**:628 (April) 1941.

22. Presented by Dr. Fred Wise before the New York Dermatological Society, Feb. 25, 1936 (Folliculitis Decalvans Associated with Keratosis Pilaris of the Trunk, Arch. Dermat. & Syph. **34**:1079 [Dec.] 1936). At the time of presentation histologic examinations were not complete.

23. Presented before the Manhattan Dermatologic Society, Oct. 11, 1938 by Dr. Isadore Rosen (Lichen Planus, Involving the Scalp, Arch. Dermat. & Syph. **39**:588 [March] 1939).

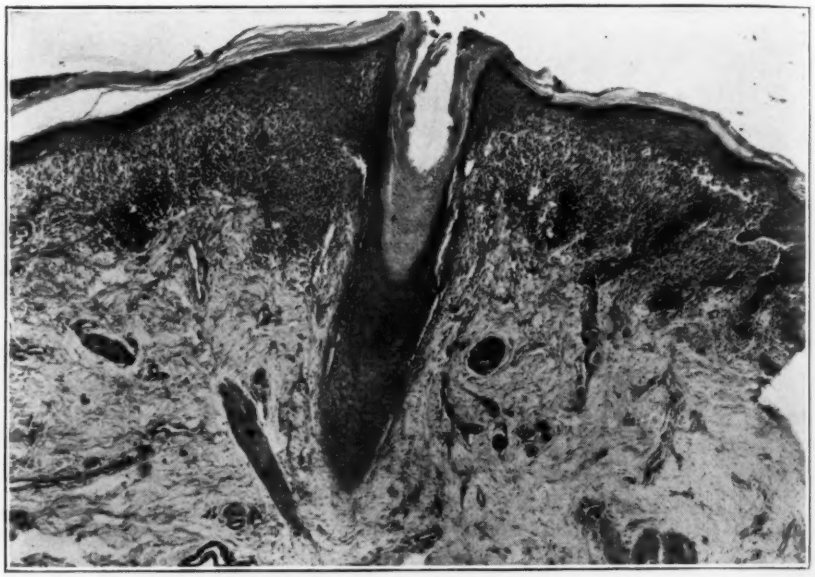


Fig. 1 (case 1).—Photomicrograph (× 53) of a section of skin from an area of alopecia on the scalp.

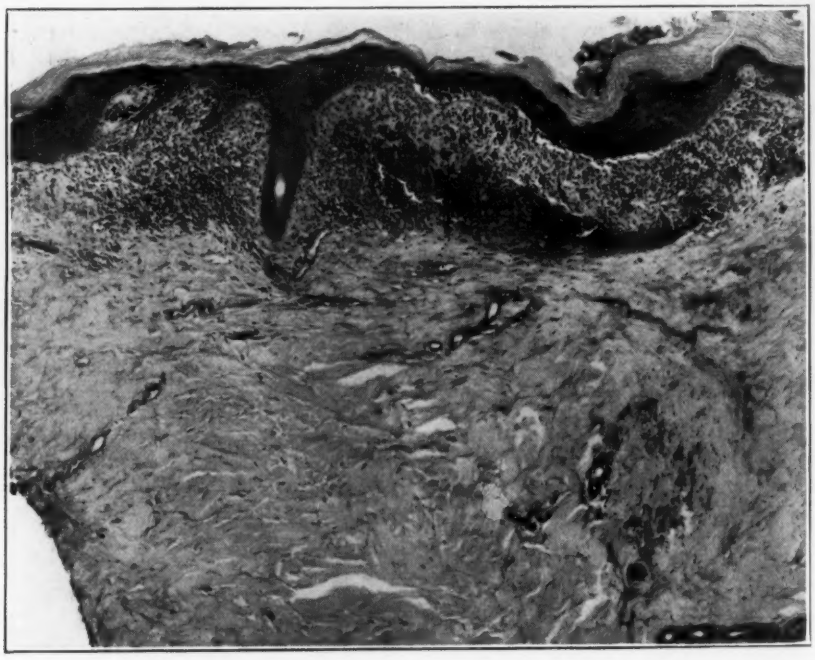


Fig. 2 (case 1).—Photomicrograph (× 18) of tissue from a lichen planus lesion involving the trunk.

was clear but for an occasional follicular plug. The hair of the axillas and pubis was not involved.

Histologic studies were made of tissue from the left temple, neck and trunk. The observations in all three specimens were essentially the same: there were hypergranulosis, platelike acanthosis and a dense band of small round cells which invaded the basal layer. Where the follicles were involved the granular layer was thickened in patches and the infiltration spread about the walls and likewise invaded the basal layer. In these sections (especially the one from the temporal plaque, which suggested atrophy clinically) the elastic tissue was missing in the area of cellular infiltration (figs. 4 and 5).

CASE 4.—A. M.,[25] a housewife aged 58, was first seen by Dr. Julius Raab in March 1939, at the Hospital for Joint Diseases, New York. She complained of a pruritic eruption of six months' duration involving the wrists, neck, trunk, legs and ankles. The lesions improved considerably under roengten therapy.

There were pinhead-sized spiny papules with central plugs scattered in groups over the shoulders, arms, abdomen and back. Covering nearly the entire vertex of the scalp was a large patch (7.5 cm. in diameter) of alopecia. The skin in this region was pinkish blue and somewhat thickened. There were no pustules at any time. At the margin were numerous violaceous follicular papules with central plugs. Surrounding the large patch of alopecia were numerous small ones (1 to 2 cm. in diameter); the skin in these areas appeared thin and suggested atrophy. In the left axilla there was a similar loss of hair.

The mucous membranes were not affected. On the wrists, abdomen and ankles were pigmented annular lesions which marked the site of the former papular eruption.

Histologic examination of a spiny follicular lesion on the shoulder showed a hyperkeratosis of the orifice. There was an increase in the granular zone in the follicle and in the adjacent epidermis. A dense band of small round cells surrounded the base of the follicle and invaded the basal margin. Elastic tissue stains showed displacement and fragmentation of elastic fibers.

CASE 5.—V. M., a 29 year old Puerto Rican woman, who has lived in the United States for the past thirteen years, was referred to the Skin and Cancer Unit by Dr. Sobel in February 1941. She complained of a pruritic eruption of three months' duration which involved the scalp, forehead, postauricular areas, neck and lumbar region.

Scattered over the neck, chest, shoulders, arms and trunk (fig. 6) were patches of acuminate, follicular keratotic papules, many of which showed central plugs and were pierced by fine lanugo hairs. In some areas the hair was lost and at its site was a white pinpoint-sized smooth spot. On the scalp were six patches of alopecia, each about 1.5 cm. in diameter (fig. 7*A*). The skin in these areas was smooth and shiny, and at the borders similar acuminate papules (such as were present on the body) were seen. The pubis and axillas were normal.

On the right buccal mucosa was a porcelain-colored area, 1 by 1.5 cm., within which were streaks and bands closely resembling lichen planus; the rest of the mucous membrane was not involved.

At the hair line on the forehead and in the temporal and postauricular regions were five pigmented nummular areas. Part of each patch was on the glabrous

25. Presented before the New York Academy of Medicine, Section of Dermatology and Syphilis, by Dr. Paul Gross for Dr. Julius Raab (Lichen Planus Spinulosus et Atrophicus, Arch. Dermat. & Syph. **43**:749 [April] 1941).

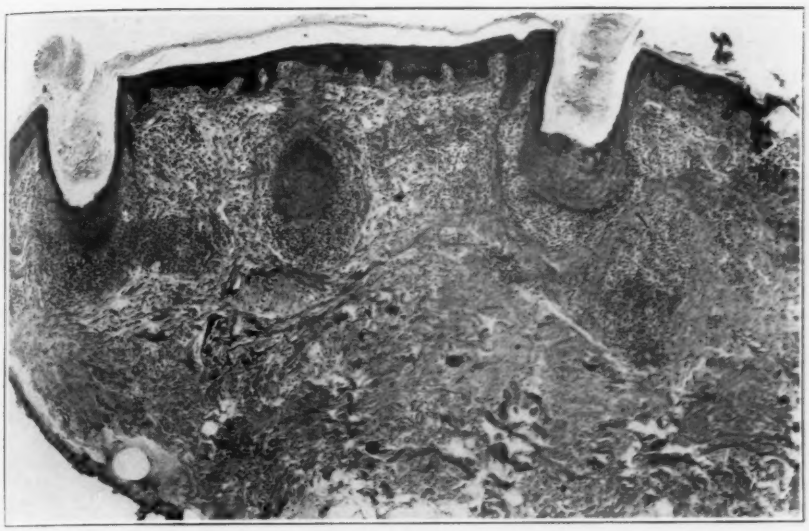


Fig. 4 (case 3).—Photomicrograph (× 43) of tissue from the scalp (fig. 3 *B*), showing the dense bandlike infiltration involving the follicles.

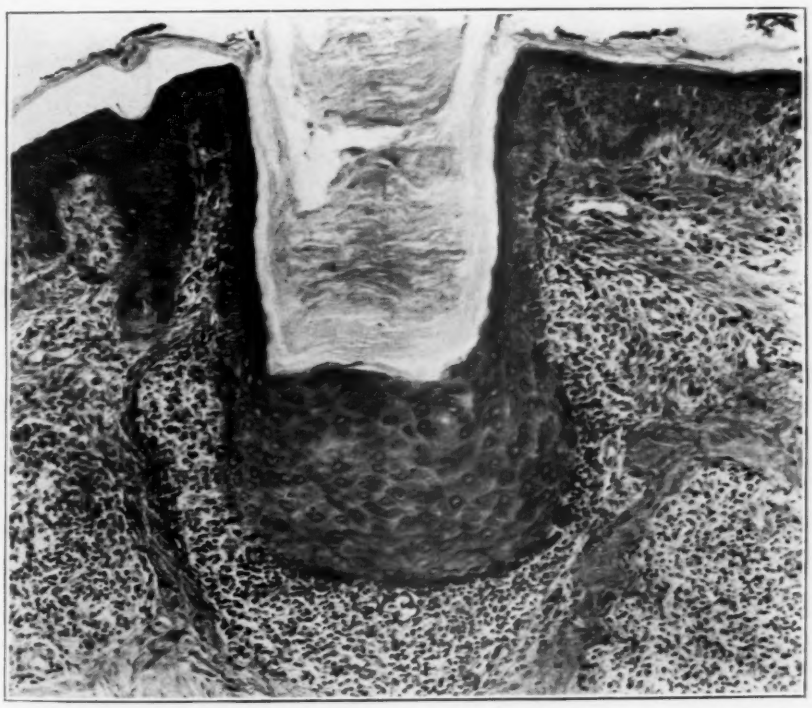


Fig. 5.—Photomicrograph (× 133) showing distortion of hair follicles by the infiltration, with obliteration of hair papilla.

skin, and the remainder extended into the scalp. This feature provided an excellent opportunity to note the effects of a single lesion on hairy and glabrous skin, showing alopecia where the hair was involved.

Skin was taken from four different areas. The histologic observations were similar: platelike acanthosis, increased granular layer and a banal infiltration which disorganized the continuity of the epidermodermal margin. In the areas showing hair follicles there was a dense band of infiltration which obliterated the hair papilla.

Fig. 6 (case 5).—Follicular lesions on the chest with follicular plugs and depressed areas in the region of the destroyed hair follicle.

Case 6.—A. F., a 34 year old Italian, was first seen at the Skin and Cancer Unit in July 1939. He complained of a moderately pruritic eruption on his face, arms and hands of two months' duration.

There was an annular pigmented lesion on each upper eyelid. On the extensor surfaces of the forearms were numerous pinhead-sized acuminate follicular papules with central horny plugs protruding from the orifices. Scattered over the forearms (but particularly on the hairy portion of the dorsa of the hands) were tiny depressions surrounding the follicles; the hair in these areas was absent, producing well defined patches of alopecia, from 1 to 3 cm. in diameter. The skin over the largest patch on the hand was slightly infiltrated and definitely violaceous.

On the right buccal mucosa was a single zone of porcelain-colored streaks and circles forming a lacelike pattern.

On the occiput were three patches of alopecia, each 3 cm. in diameter. The skin in these areas, however, was smooth and normal in appearance. The follicular papules seen elsewhere on the body were not present here (fig. 7 *B*).

Skin was taken from the eyelid, elbow, dorsum of the hand and buccal mucosa. The histologic changes were similar, showing the characteristic changes seen in lichen planus. Sections from the hairy regions (forearm and hand) showed a dense

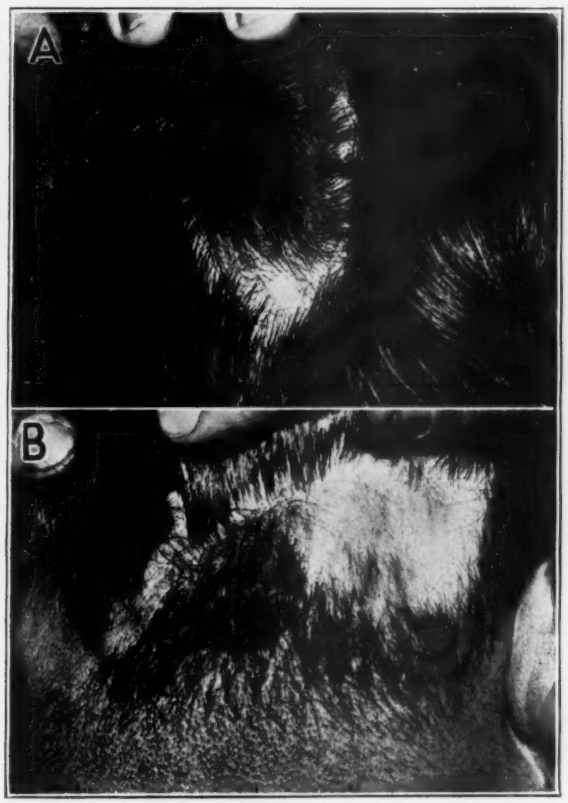


Fig. 7—Alopecia of the scalp. *A*, case 5. *B*, case 6.

small round cell infiltration invading the basal margin of the follicles and distorting their contour by pressure.

DIFFERENTIAL DIAGNOSIS

A review of the literature disclosed the various names which have been given to this entity: In spite of the fact that Little had retracted his original title of lichen spinulosus with folliculitis decalvans, many subsequent authors (Dore, Beatty, Ormsby, Senear, Traub and Tolmach) reported cases under this name.

That the process is not folliculitis decalvans seems generally accepted. Follicular pustules, the essential feature of the disease described by

Quinquaud,[26] are never present. We were able to find only 1 case in which there were pustules (presented by Senear [27]). Possibly the condition in this case was not lichen planopilaris but folliculitis decalvans.

The greatest difficulty has been in appraising the acuminate follicular lesion found in lichen planopilaris. This has been confused with keratosis pilaris, keratosis follicularis, lichen spinulosus and pityriasis rubra pilaris.

In keratosis pilaris the scalp as well as the lateral aspects of the arms and thighs may be involved. However, alopecia is never part of this persistent disease. Further, it is generally accepted that keratosis pilaris is not an inflammatory but rather a nevoid dermatosis. Thus far, the work with vitamins has not disproved this, nor has the tuberculid theory been seriously entertained in this country.

Lichen spinulosus (lichen pilaris seu spinulosus) is a disease of childhood which is persistent and may last indefinitely. It has been suggested by Adamson that lichen spinulosus in the adult ought to be regarded as a variety of lichen planus. We partly agree with this but believe that some of the follicular keratoses possibly belong in the group of avitaminoses and probably are in no way related to lichen planus.

It is true that the individual lesions of lichen planopilaris and pityriasis rubra pilaris resemble each other. In none of the cases, however, has the classic picture of the latter disease been seen. No instance of general eruption has been reported. The characteristic palmar and plantar thickening as well as other essential symptoms are not present in the follicular type of lichen planus. The individual lesion is somewhat similar, but in no other way is there any resemblance to the picture of keratosis follicularis.

In addition to the clinical differences, the microscopic observations are of value. The characteristic changes found in the aforementioned diseases are distinct from those seen in the process under consideration. This will be considered in another part of the paper. For the present suffice it to say that the follicular, as well as the planus, variety of lichen planus has a characteristic histologic picture which can be differentiated from keratosis pilaris, keratosis follicularis and pityriasis rubra pilaris.

CLINICAL FEATURES

Lichen planopilaris is usually an insidious process. Most of the patients give a history of gradual involvement over a period of several months (or even one or two years). In some the scalp may be affected first, and in others the glabrous skin. Subjective symptoms are usually mild; however, 1 of our patients complained of severe pruritus.

---

26. Quinquaud, E.: Folliculite destructive des régions vélues, Bull. et mém. Soc. méd. d. hôp. de Paris **5**:395, 1888.

27. Senear, F. E.: Folliculitis Decalvans and Lichen Spinulosus: A Clinical Combination, Arch. Dermat. & Syph. **2**:198 (Aug.) 1920.

On the scalp (or other hairy parts of the body which may be affected) usually are two or three, or even as many as ten to twelve, patches of alopecia, varying from 0.5 to 3 cm. in diameter. Occasionally they may coalesce so that a palm-sized area results. The skin of the involved area usually shows a varying degree of thickening; the color may be normal, slightly erythematous or violaceous. Typical papules of lichen planus are not usually seen on the scalp; in 1 of our cases (case 2), however, there were flat-topped shiny violaceous papules in this area. In the majority of cases there are acuminate follicular lesions scattered over the involved area but more numerous at the margins of the alopecia. An almost constant observation is the presence of minute keratotic plugs and pinpoint-sized shiny depressions marking the sites of the follicles.

On the glabrous skin the lesions are of two types: 1. Typical papules of lichen planus were present in 4 of our 6 cases. When present they are found in the usual locations (neck, wrists and ankles). Associated with these, involvement of the mucous membrane may be noted. It is interesting that annular pigmented lichen planus, a relatively uncommon lesion, was present in 4 of our cases. 2. All the patients we observed had follicular lesions on the body. Where adult type of hair was present (on the chest, face and arms, as well as in the axillas and on the abdomen and pubis) alopecia was more evident. When hair on the body was involved, the picture was similar to that seen on the scalp. The pinpoint-sized shiny depressions (which we have already described as occurring on the scalp) were consistently found on the body, though they were not so prominent where the lanugo hairs were situated. The areas of predilection for the second type of lesion are the neck, shoulders, chest and extensor surfaces of the arms and legs, though any part of the glabrous skin may be so affected.

In our series of patients 3 were males and 3 females. Their ages varied from 24 to 58 years. The duration of the process when first seen by us varied from one month to a maximum of one year; three to four months was the average.

Various types of treatment, including arsenicals, bismuth, vitamins—particularly large doses of vitamin A (50,000 to 100,000 U. S. P. units daily)—local applications and roentgen rays, did not influence the alopecia. The follicular lesions improved slowly with either injections of a heavy metal or roentgen therapy.

MICROSCOPIC OBSERVATIONS

The histologic changes in sixteen specimens, taken from 6 patients, were consistent.

Sections of the flat and of the follicular type of lesion showed the same basic picture. An increase in the granular zone, platelike acanthosis, a well defined band of small round cells and dissolution of the basal margin (observations which make the microscopic picture of lichen planus char-

acteristic) were seen in all of our specimens. Several sections showed Max Joseph's lacunas.

The interesting feature was in connection with the process as it affected the hair follicles. All the essential changes seen in lichen planus were found where the follicles were involved: hypergranulosis, acanthosis and a well defined zone of small round cells which invaded the basal margin. In addition, there were alterations of the normal structure of the follicle (more particularly the papillary portion). The infiltration uniformly pressed against the base of the follicle so that the papilla was obliterated. As the source of blood supply to the hair bulb comes through the hair papilla, it may be that the pressure was sufficient to interfere with the nutrition of the hair bulb and thus produce ischemia and the eventual loss of that structure. This mechanical interference with the blood supply may account for the loss of hair.

Elastic tissue stains of these sections and of sections from 10 cases of unquestionable lichen planus, for purpose of control, showed absence of elastic fibers within the infiltration and some clumping and fragmentation at the periphery and particularly in the neighborhood of the follicles. Such changes in the elastic tissue are not indicative of atrophy.

COMMENT

With this accumulation of cases it appears highly improbable that the two conditions (lichen spinulosus and folliculitis decalvans) would occur together so often as a coincidence. With our 6 cases, a total of 27 now have been described in the American literature.

It is not our purpose to attempt to add anything to the observations of Pringle, Adamson, Fox and Fordyce concerning follicular lichen planus. We are primarily interested in gathering together a fairly large series of cases called by various names but having essential features in common. We wish to show that this variously titled entity is a follicular variety of lichen planus. In our opinion it is and was unnecessary to coin a new term to describe it; the one introduced by Pringle, lichen planopilaris, is a proper and adequate one.

Feldman entitled this dermatosis lichen planus et acuminatus atrophicans, and most subsequent cases reported by others bear this designation. This title is partly correct in that it places this dermatosis in the lichen planus category; however, it does not suggest the essential feature, namely, follicular involvement, and it allows for possible confusion with lichen acuminatus (pityriasis rubra pilaris). For these reasons we feel that the term lichen planopilaris (Pringle) is preferable.

The term "atrophicans" may be only a clinical assumption. There are cases in which the lesions do not suggest atrophy even clinically. However, there is little doubt that often the lesions do appear to be atrophic, as may be noted in clinical descriptions of our cases and of other

reported cases. The only proof thus far of atrophy is the change noted in the elastic tissue. However, this cha ge is identical with that seen in lesions of lichen planus, in which even linical atrophy is not observed.

Examination of tissue from clinically atrophic areas on the scalp still showed active follicular lichen planus with changes in the elastic tissue similar to those described. It is obvious from what has been said before that the hair is permanently missing in areas where the follicles are involved. Thus, it may be argued that this is atrophy. However, we believe that this is not the implication given by Feldman, who stated that the lesions both of lichen planus and of the follicular variety go on to atrophy.

In 4 of the reported cases and in 3 of our own the diagnosis of pseudopelade was considered, as the clinical picture was no different from other accepted cases of that disease that we have observed. If the scalp alone were involved or if one had overlooked the glabrous lesions, the diagnosis of pseudopelade could have been acceptable. This brings up a question: Is it possible that pseudopelade is not an entity but a manifestation of lichen planopilaris? The histologic changes of pseudopelade, as described by Photinos [28] in a comprehensive study of the subject, show some similarity to those seen in our cases of alopecia resulting from lichen planopilaris.

SUMMARY AND CONCLUSIONS

The pertinent literature has been reviewed and 6 additional cases have been recorded to show that Little's disease and lichen planus et acuminatus atrophicans (Feldman) are the same as lichen planopilaris, described by Pringle.

We agree that some cases of lichen spinulosus in the adult should be regarded as instances of follicular lichen planus (lichen planopilaris), as first suggested by Pringle and Adamson.

Lesions of alopecia are not necessarily confined to the scalp but may be noted on other hairy parts.

The application of the term atrophy to this dermatosis is based on a clinical assumption and not on a proved histologic fact and warrants further investigation.

Further observations might prove whether or not it is correct to assume that pseudopelade, at least in some cases, may be the result of lichen planopilaris of the scalp.

Drs. Julius Raab and N. Sobel gave us permission to include their cases in our series.

921 Bergen Avenue, Jersey City, N. J.

3164 Meadowbrook Road, Cleveland Heights, Ohio.

28. Photinos, P. B.: La pseudo-pelade de Brocq, Paris, N. Maloine, 1930.

ELECTROLYSIS

INTRODUCTION OF AN INSTRUMENT FOR RELATIVELY PAINLESS TREATMENT

EUGENE A. HAND, M.D.
SAGINAW, MICH.

The destruction of the hair follicle in congenital dystrichiasis and in trichiasis associated with trachoma and other pathologic conditions of the eyelids causing entropion has long been an ophthalmologic problem. Michaels,[1] an ophthalmologist, in 1869 first introduced the method of inserting a needle heated to white heat into the follicle to destroy it. This was effective but painful and dangerous. In 1875 he perfected and used with success the negative pole electrolysis instrument.

In 1878, at one of the first meetings of the American Dermatological Association, Bulkley [2] suggested the rather barbaric method for treating hypertrichosis of inserting a razor sharp, two-edged needle into the follicle. This was then twisted to destroy the follicle. Often the needle was to be dipped into phenol first. Duhring,[3] at the same meeting, suggested, perhaps facetiously, a triangular needle for the same purpose.

These methods were not popular, and the dermatologists rapidly took up the method and instrument perfected by Michaels.

Duhring,[4] in 1881, designed the electrolysis handle used today.

Rosenberg,[5] in 1925, suggested the multiple needle method. He stated the belief that this method was faster and less painful. The method has been popular with the lay operator and cosmetician but never with the dermatologist.

High frequency diathermy for destruction of the follicle was first suggested by Bordier,[6] in 1932. The slowness of its general acceptance was due, according to Karp,[7] to the fact that the diathermy units in most dermatologists' hands are not suited to or adjusted for this use.

This paper was presented before the Section on Dermatology and Syphilology at the meeting of the Michigan State Medical Society at Grand Rapids, Mich., in September 1939.

1. Michaels, C. E.: St. Louis Clin. Rec. **2**:145, 1875.
2. Bulkley, L. D.: Arch. Dermat. **4**:287, 1878.
3. Duhring, L. A., in discussion on Bulkley.[2]
4. Duhring, L. A.: Am. J. M. Sc. **83**:142, 1881.
5. Rosenberg, W.: Illinois M. J. **68**:138, 1925.
6. Bordier, D. H.: Am. J. Phys. Therap. **8**:273, 1932.
7. Karp, F. L.: High Frequency Current in Treatment of Hypertrichosis, Arch. Dermat. & Syph. **43**:85 (Jan.) 1941.

In recent months I have used this method extensively for treating hypertrichosis. It is speedy and most effective and will most likely supersede electrolysis for treatment of this condition. The percentage of permanent epilation in my hands has been much higher with this method.

The electrolysis instrument and technic perfected by Michaels were borrowed and used by the dermatologists without any improvement or change. Besides using them for hypertrichosis, the dermatologists have used them for the removal of small benign growths, such as nevi, verrucae, molluscum contagiosum, adenoma sebaceum, milia and keloids. This is the method par excellence for treatment of telangiectases and small hemangiomas. Electrolysis has even been tried as an irritant in conjunction with croton oil or trichloroacetic acid to set up a kerion-like inflammation in simple tinea of the scalp.

Unfortunately, the method is not painless. This fact has greatly limited its use. The purpose of this paper is to present an improved electrolysis instrument which in actual practice causes much less pain, though it is just as effective and useful.

THE MICHAELS METHOD AND AN ANALYSIS OF THE PAIN INCIDENTAL TO ITS USE

Figure 1 illustrates the circuit used in the Michaels instrument and also the pain incidental to its use. When the make and break switch is closed, the galvanic current rises instantaneously from 0 to maximum with a definite painful shock. As the current flows around the circuit, the electrolytic action at the negative pole or needle proceeds with a burning sensation which gradually decreases as the cells and nerve receptors about the needle are destroyed. When the circuit is broken, the current drops again instantaneously to 0 with a shock somewhat less painful than the make shock, owing to the partial anesthesia about the needle.

The pain of the electrolysis procedure can be lessened or eliminated either by the use of a topical or local anesthesia or by an improvement of the electrolysis instrument. Topical anesthesia on the skin is not satisfactory. Local anesthesia prior to electrolysis for numerous superfluous hairs is not practical. Its use prior to treating telangiectases causes the capillaries to fade, making the electrolysis a hit or miss affair. When its use is practical, as in the removal of a few nevi or verrucae, it should be employed.

The pain with the Michaels circuit can be divided into its two components. The useful component is that burning pain associated with the electrolytic action on the tissues. The useless component is that caused by the make and break shocks. The useful component cannot be

eliminated entirely. It can be minimized by using a maximum current of 1 milliampere. As the burning pain decreases in the performance of electrolysis, a method to raise the maximum gradually to utilize this partial numbing will further minimize the pain. This can be easily done with the instrument to be presented.

The pain incidental to the make and break shocks, as well as the shocks themselves, is useless. From the reaction of patients treated with my first instrument copied after the Michaels circuit, I learned that this was what they dreaded. The pain was not so severe, but they feared its constant recurrence. This was much the same reaction that many medical

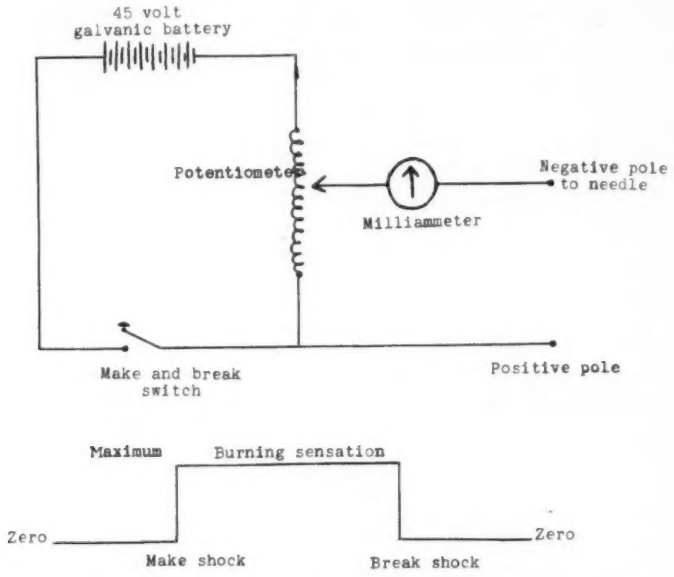


Fig. 1.—Diagram of Michael's circuit and pain incidental to its use.

students have to jabbing their finger to obtain blood for learning to make blood counts. The first few jabs with the blood stylet are easy, but soon the fear of the slight pain makes it almost impossible for the student to jab the skin. This useless pain of the electrolysis procedure can be entirely eliminated by gradually raising and lowering the current from 0 to maximum and then back to 0 to eliminate the make and break shocks. The simplicity of this method surprised me. Rosenberg [5] was close to this principle when he stated that the multiple method was less painful because the make and break shocks were less frequent. Menaugh [8] also utilized this, as he had a nurse gradually raise and lower the current by hand manipulation of the potentiometer.

8. Menaugh, F.: Personal communication to the author, 1939.

METHODS OF GRADUALLY RAISING AND LOWERING THE CURRENT

The current can be raised and lowered in a number of ways. The following methods were tried in the order given before the foot-controlled potentiometer was devised:

1. Hand manipulation of the potentiometer was early discarded, as it required the help of an assistant if the operator's hands were to be free for action. This complicated a simple procedure.

2. A rectifier tube controlled by a make and break switch inserted in the Michaels circuit in place of the make and break switch will

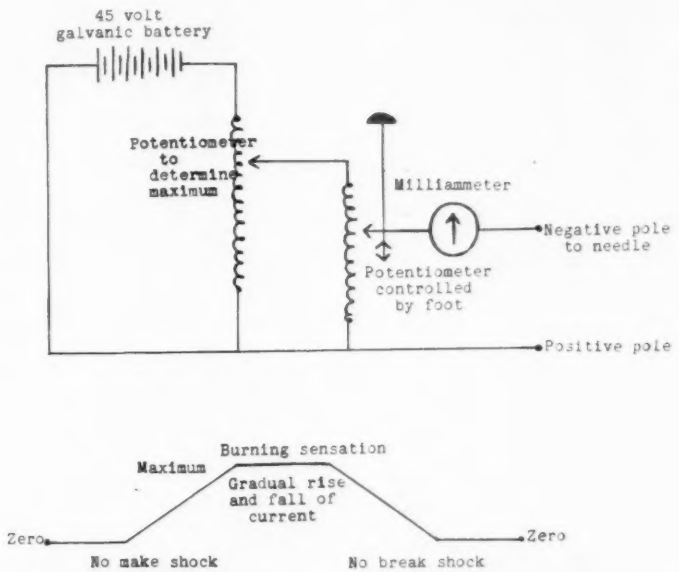


Fig. 2.—Diagram of circuit used in improved electrolysis instrument.

eliminate the shocks and their associated pain. When the circuit for the rectifier tube is closed, the tube slowly heats up, allowing more and more electrons to flow around the electrolysis circuit till the maximum is reached. The reverse happens when the tube cools. The maximum current is not under easy control by the operator, nor is the rate of the rise and fall of the current under his control; these are the disadvantages of this method.

3. The foot-controlled potentiometer is used in the instrument presented. This is a modification of the hand-controlled instrument. The make and break switch in the Michaels circuit is replaced by another potentiometer. This is attached by a gear and rack system to a foot pedal. Pressure on this pedal raises the current, and removal of this pressure lowers the current. Figure 2 illustrates this circuit with the

completed instrument. With this instrument, the rise and fall of the current and the maximum at any time are easily controlled by the operator's foot.

TECHNIC

The patient is placed on a table with the part to be treated toward and below the operator. The latter is seated on a comfortable chair or

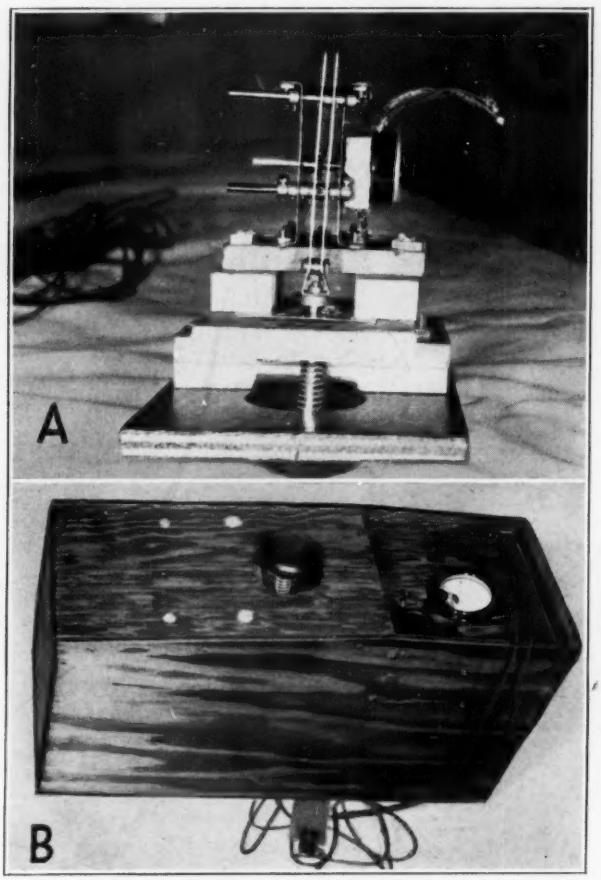


Fig. 3.—*A,* inside of the electrolysis instrument, showing the foot pedal and rack and gear system. *B,* the finished instrument, showing the upper surface.

stool with the instrument on the floor so that the foot pedal can be easily reached.

The positive or indifferent electrode is held by the patient or strapped to his body. With the foot pedal relaxed, the negative pole or needle is inserted into the follicle or part to be treated. The foot pedal is slowly

depressed, raising the current. A close watch of the patient's facial expression to note pain guides the operator. The pedal is gradually raised as soon as the desired electrolytic action has occurred, at which time the needle can be withdrawn. The procedure can be repeated at will.

With this method, the make and break shock and the associated pain are entirely eliminated. The burning pain from the electrolysis is minimized by gradually raising the current maximum to take advantage of partial anesthesia from the destruction of the pain receptors about the needle.

4. The iontophoresis instrument presented by Haggard, Strauss and Greenberg [9] can be and is used for electrolysis. With this the current is raised and lowered by means of condensors and choke coils. Though I have had no experience with this instrument, it is my belief that the speed of the rise and fall of the current would not be easily controlled by the operator, if at all.

THE USE OF THE INSTRUMENT PRESENTED AS A PULP TESTER

The technic for the use of this instrument as a pulp tester is as follows: The positive pole is held by the patient or strapped to his body. The negative pole or needle is placed on the tooth adjacent to the nerve, as with other pulp testers. The foot pedal is then slowly depressed till the patient feels the current flowing. The reading on the potentiometer is then taken at this point and compared with similar readings from known live, dead and hypersensitive teeth. From this comparison the state of the tested tooth can easily be determined.

SUMMARY AND CONCLUSIONS

The electrolysis instrument perfected by Michaels in 1875 is essentially the same as that used today. The pain incidental to its use has prevented it from being popular. This pain consists of two components:

The useful component is that due to the electrolytic action on the tissues. This can be decreased by using a small maximum milliamperage and also by gradually raising the current to take advantage of the partial anesthesia about the needle as the cells and nerve receptors are destroyed.

9. Haggard, H. W.; Strauss, M. J., and Greenberg, L. A.: Fungous Infections of Hands and Feet Treated by Iontophoresis of Copper: Preliminary Report, J. A. M. A. **112**:1229 (April 1) 1939.

The useless component can be entirely eliminated by gradually raising and lowering the current to eliminate the painful shocks caused by the sudden make and break of the current which is found with the Michaels circuit.

A foot-controlled instrument to eliminate these shocks and their associated pain and also to facilitate the gradual raising of the current is presented.

This instrument can also be used by the dentist as a pulp tester to test the vitality of teeth.

801 Second National Bank Building.

RATIONAL PRESCRIPTION WRITING IN DERMATOLOGY

HERMAN GOODMAN, M.D.
NEW YORK

It has been common for many years to decry the made-to-order prescription suggestions for the treatment of diseases of the skin. Active medicaments are advised, but the mode of application is ignored. For the most part, physicians recall the pharmacology of cutaneous applications sufficiently to take advantage of the reducing agency of a large number of drugs. The theories of the late Paul Unna may not satisfy all the requirements of modern reduction-oxygenation ideas. However, they retain sufficient clinical responses to be worth remembering. In this presentation, little will be said of active agents. The response to be anticipated by the mode of application will be stressed.

CREAMS

The dermatologist rarely intentionally prescribes or orders a pharmacist to prepare a cream with U. S. P. and N. F. ingredients. The reason is that patented medicaments and chemicals may not be included in these official lists. Modern cosmetic creams available for physicians and pharmacists are made with patented material. Creams may be defined as mixtures of fat and fatlike substances bound with water through physical, chemophysical or chemical intermediaries, or mixtures of them. In an older day of cream making, certain physiochemical components of hydrous wool fat were utilized. Thus, wool fat and hydrous wool fat are well known. Soap was another favorite medium for binding fat and fatlike substances with water.

Hydrous wool fat emulsions (an emulsion is defined as a close union of otherwise immiscible fat and fatlike substances with water) are still popular. They have a decided field of application in dermatology. The intermediary is recognized as one or another of the oxycholesterol quotients of hydrous wool fat. Soap-bound creams are ordinarily made through monovalent metallic salts (sodium, potassium or ammonium) and fatty acid through saponification (defined as the formation of soap within the formulation of the cream). The saponified cream is unable to tolerate electrolytes and hence is a poor medium or vehicle for active medicaments.

The art and science of making creams creates emulsions through mechanical agitation. Intermediaries may or may not be added. Pectin

has been included in the list of physical intermediaries or stabilizers for dermatologic creams. The sulfonated oils are valuable binders of the chemophysical class. Diglycol laurate and diglycol stearate are new chemicals available for the chemical binding of otherwise immiscible fat and fatlike substances and water.

Emulsions are classified according to whether they are oil in water or water in oil emulsions. They are either thick (creams) or thin (milk or lait). Emulsions are also divided into temporary (not desirable) and permanent types.

The higher alcohols, cetyl, stearyl, octyl and their combinations, are increasingly important ingredients of greases and more especially of creams. Cetyl alcohol may well be added to any pomade or grease or its water-added counterpart, cream. The addition of no more than 2 per cent improves any such application for the skin, improves the appearance of the finished product and improves the appearance of the skin at the site of application. The cetyl alcohol component causes the skin to assume a velvety sheen and feel. The cetyl alcohol assists in combining fat and fatlike materials and water through a physical binding power. Cetyl alcohol is commercially available under many guises and in various percentages of purity to 99 per cent at increasingly higher cost. It is essential to have the purveyor present an analysis of the product purchased with a statement of its physical properties.

Stearyl and octyl alcohols are available for the most part in commercial combinations. Although the trade names may remain unchanged, it is possible that the ingredients or their proportions may vary in different samples or lots. The higher alcohols in solid form present unusual opportunities for experimentation in formulation of pomades and creams with the advantage of acting as if completely absorbed by the skin or the scalp. Creams prepared with combinations of these solid alcohols are water removable without agitation. The injured skin or scalp is thoroughly and rapidly cleansed without the addition of irritation through the detergents required for the removal of conventional fat or cream bases. The higher alcohols in bases for creams permit the incorporation of electrolytes without destroying the emulsion-like characteristics of the cream vehicle.

The inactivity to p_H of creams prepared with glyceryl monostearate brings this product to the attention of dermatologists. The finished cream is easily made, and almost no attention need be paid to incompatibility. Acid and alkaline remedies may be added without breaking the emulsion.

Results from the application of creams prepared with these higher alcohols have been uniformly excellent. They should be freely prescribed as soon as they are generally available in pharmacies.

Sulfonated oils, such as sulfonated castor oil, sulfonated olive oil and sulfonated coconut oil, are available in various degrees of sulfonation. These make ideal soapless shampoo ingredients and are rarely the cause of any untoward reaction. The sulfonated oils are readily miscible with water. They may be dispensed diluted or modified by the addition of liquid petrolatum. Sulfonated oils disperse readily; hence the sulfonated castor oil, for example, is a good menstruum for medicaments intended for the bath.

The mention of liquid petrolatum suggests the need of insisting at this place that neither the light nor the heavy liquid petrolatum of the United States Pharmacopeia should be compounded for cutaneous applications. A low viscosity liquid petrolatum, with a viscosity between 55 and 80, is recommended for incorporation in prescriptions intended for the skin or scalp.

The trade names for creams, such as shaving cream, cleansing cream, vanishing cream, brushless shaving cream and cold cream, do not indicate the mode of preparation or the ingredients. The cold cream given in the United States Pharmacopeia, rose water ointment, is a finely balanced, relatively expensive, permanent emulsion of the thick type, intolerant of active electrolytes. A pharmacist knowing its weakness does not mix salicylic acid with it, for example. If the prescriber designates this particular ointment, the pharmacist substitutes a nonofficial cream, with or without consent for this combination. A physician blames the pharmacist who follows his order for a poor salicylic acid cream and lauds the pharmacist who has made a substitution.

The following formulas for cream are suggested:

SOFT TRIETHANOLAMINE SOAP CREAM

Stearic acid	4 Gm.
Hydrous wool fat	4 Gm.
Cetyl alcohol	2 Gm.
Liquid petrolatum	4 cc.
Corn oil	4 cc.
Triethanolamine	1 Gm.
Water, enough to make	100 Gm.

Melt the solids in mixture of warmed oils, and stir with warmed water into which the triethanolamine has been dissolved.

VANISHING CREAM BASE

Glyceryl monostearate	12 Gm.
Yellow wax U. S. P.	2 Gm.
Glycerin	6 cc.
Corn oil	4 cc.
Water, enough to make	100 Gm.

Heat together until solids are melted, and stir to form cream.

HAND CREAM

Glyceryl monostearate	16 Gm.
Hydrous wool fat	4 Gm.
Cetyl alcohol	1 Gm.
Liquid petrolatum	4 cc.
Cottonseed oil	4 cc.
Corn oil	4 cc.
Water, enough to make	100 Gm.

Melt solids in mixture of warmed oil, and stir with solution of glyceryl monostearate in hot water.

CLEANSING CREAM

Yellow wax U. S. P.	4.0 Gm.
Stearic acid	0.6 Gm.
Sesame oil	60.0 cc.
Corn oil	2.0 cc.
Cottonseed oil	2.0 cc.
Hydrogenated oil	12.0 Gm.
Sodium borate	0.6 Gm.
Water, enough to make	100.00 Gm.

Melt the solids; add the warmed liquids, and mix with solution of sodium borate in water warmed in separate container.

GREASES

Pomades or greases, also known as ointments and including bases for pastes, may be defined as mixtures of fats and fatlike substances physically bound together, essentially free of water. Greases may include other ingredients, medicaments or modifiers conveying activity, depending on whether they are inert or active. Greases or ointments to be effective on the skin must have melting points approximating the temperature of the skin at the site of their application. Greases engender heat and cause congestion. They adhere to the part, prevent evaporation of fluids and afford physical protection.

In earlier practice, animal fats predominated in grease bases. Lard and hydrous wool fat appeared in the formularies. Lard has been replaced in official ointments during the past decade. The oxycholesterol quotients are utilized instead of the hydrous wool fat. Mineral greases and oils prevail. Amber, white and liquid petrolatum are popular.

The conventional ideas and concepts of an ointment base called endodermic, as distinguished from epidermic, are accepted. The former has a vehicle intended to permit activity within the skin. The latter (epidermic or superficial) has a vehicle intended to limit the activity to the surface of the skin. To complete the picture, one includes the diadermic ointment base, assumed to permit or assist the passage of medicaments through the skin.

There is much controversy regarding the actual limitations or possibilities of these classes of ointment bases. The available evidence is far from convincing. All of it purporting to prove the possibility of

penetration or absorption through the healthy unbroken epidermis should be reviewed and repeated by impartial, competent laboratory investigators with significant control experiments. This doubt does not prohibit giving definitions.

Epidermic ointment bases do not have much, if any, reputation for permitting penetration of included medicaments into the skin. Mineral oil (liquid petrolatum), mineral greases and mineral waxes are ingredients of epidermic ointments. Combinations of mineral greases offer fairly low melting points, at least a few degrees below the temperature of the mouth.

Endodermic ointment bases have the reputation of permitting penetration of included medicaments into the depth of the skin. Lard, wool fat and hydrous wool fat have the reputation of being valuable as bases for endodermic ointments. They offer certain definite objections as sole ingredients.

Diadermic ointments have the reputation of permitting penetration of included medicaments through the skin into the organism. Certain volatile drugs enter the blood stream and give measurable effects on application in a diadermic ointment base. The mode of entry may have been through the air passages rather than through the intact skin. Goose grease was offered as a vehicle for diadermic ointments in the early days of pharmacy. Few city pharmacies stock it nowadays.

Space does not permit a discussion here of the biologic or chemical activities in the field of emulsification of animal grease ointment bases as contrasted with mineral grease ointment bases. A definite loss has been sustained by the acceptance of mineral fats and fatlike substances for the animal fats.

Hydrogenated fats are available on the market with all degrees of hydrogenation. The commercial household hydrogenated oils and fats, recognized as shortening and used as substitutes for butter in frying, baking and other cooking, are available for grease, ointment and paste vehicles. There is a modicum of nickel catalyst present, but this does not interfere with the finished ointment-like product. Individual experimentation will be required for each trademarked hydrogenated oil or fat preparation to determine the percentages needed for specific melting point combinations with oils and other fats.

Cerates are combinations of fatty bases and animal waxes not soft enough to melt and run when applied to the skin.

Pastes are ointment-like mixtures of powders with equal parts of glycerin, soft soap, petrolatum, lard or some other fat or fatlike combination or mixture. Pastes are protective; they act to soften the surface they cover, yet they are more or less porous, being permeable to perspiration, and act to relieve congestion. Bentonite is a good carrier for chemical reducing action in pastes.

A former popular prescription for a paste included 25 per cent of starch. Nearly all formularies have this prescription, and many physicians order it. The type of starch incorporated in the product is of great importance. Most pharmacists, including those of large dermatologic clinics and hospitals, mix dry starch with the other powders and agitate into the fat menstruum. A more desirable and rational product is formed if the starch is first hydrolyzed by agitation in cold water, followed by heating with constant stirring to form a paste.

The following formulas for greases are suggested:

SULFUR OINTMENT

Washed sulfur	20 Gm.
Potassium carbonate	10 Gm.
Water	10 cc.
Wool fat	10 Gm.
Hydrogenated oil, enough to make	100 Gm.

Dissolve the potassium carbonate in water; incorporate with wool fat, and mix intimately with powdered sulfur and hydrogenated oil.

MODIFIED STARCH AND ZINC PASTE

Starch	25 Gm.
Water	50 cc.

Rub until smooth; heat with constant stirring to form paste.

Zinc oxide	25 Gm.
Liquid petrolatum	25 cc.
Hydrous wool fat	25 Gm.

Mix.

OINTMENT BASE

Solid alcohol (cetyl-stearyl), self emulsifiable	30 Gm.
Hydrous wool fat or petrolatum	30 Gm.
Water, enough to make	100 Gm.

Melt the solids, and mix with warmed water, stirring to cream.

SKIN PEEL (SUPERFICIAL) PASTE

Resorcinol	40 Gm.
Zinc oxide	10 Gm.
Bentonite	6 Gm.
White petrolatum, enough to make	100 Gm.

Mix the powders, and incorporate into the grease.

SCALP POMADE

Resorcinol monoacetate	4 Gm.
Castor oil	30 cc.
Sesame oil	30 cc.
Cetyl alcohol	2 Gm.
Cerisin	20 Gm.
Hydrogenated oil and Petrolatum, of each enough to make	100 Gm.

SOLUTIONS

Solids, liquids and gases completely dispersed in fluid mediums are recognized as solutions. Solutions present pharmacologic opportunity for the dermatologist according to the fluid medium and according to the material dissolved therein. Aqueous solutions of volatile substances are called waters; solutions of nonvolatile substances are called liquors, and tinctures are alcoholic or hydroalcoholic solutions of active principles of animal or vegetable drugs or chemicals. A favorite tincture in dermatology is tincture of iodine. It is offered for some phases of the common cutaneous affliction, tinea of the toes. A variation of a diluted tincture of iodine with the addition of tincture of aconite is therapeutically more efficient than the tincture of iodine alone. The combination must be applied with some caution, as a few persons are sensitive to aconite. For the most part, solutions, particularly aqueous ones, are cleansing, relieve congestion, allay irritation and soften the corneum irrespective of the materials in solution.

Industrial research has offered the medical art many new solvents. These require much study, and few have been accepted by the official pharmaceutic groups. The possibilities of toxic reactions from the host of glycols must not be forgotten. Physicians who prescribe must be doubly cautious, since they treat the abnormal or diseased skin. The cosmetician and the beauty chemist assume risks that physicians must avoid. Theoretically, the beautician dispenses for the normal, healthy and unbroken surface of the skin.

Ethyl alcohol and isopropyl alcohol are available as liquid vehicles either alone or with water (hydroalcoholic) or with mixtures of glycerin and water. Evaporation of alcohol is cooling in sensation and in effect. Its action is rapid, hence short in duration for each application.

One solution has been miscalled a lotion for upward of six decades. Lotio alba, or white lotion N. F., has fallen into disfavor because of this error. As properly written and prepared, white lotion (to retain its popular but misleading name) is actually a solution of hydrogen sulfides in water with a suspension of insoluble precipitate resulting from the interaction of the aqueous solutions of its ingredients (acid zinc sulfate and alkaline sulfurated potash—not potassium sulfide).

The active ingredient of the so-called white lotion is actually a gas having the characteristics of a reducing agent in the sense of Unna. The gas is in solution in the supernatant liquid of the "lotion" and also held in the precipitate. The preparation known as solid lotio alba, made by the interaction of a high concentration of zinc sulfate and sulfurated potash, is pharmacologically unsound. The gas evolved passes into the atmosphere of the pharmacy. Only the portion of the gas captured physically by the otherwise inert precipitate is temporarily available to

act on the skin. The addition of a powder or the decantation of the supernatant fluid to prepare "solid lotio alba" is sophistry and denotes ignorance of the mode of activity of the preparation.

The following formulas for solutions are suggested:

TINCTURE OF IODINE AND ACONITE DILUTED

Tincture of iodine	10 cc.
Tincture of aconite	10 cc.
Alcohol, enough to make	100 cc.

Mix.

SCALP WASH

Quinine bisulfate	1 Gm.
Beta naphthol	4 Gm.
Sulfonated castor oil	12 cc.
Isopropyl alcohol	50 cc.
Water, enough to make	100 cc.

Mix.

TWO BOTTLE "WHITE LOTION"

1. Zinc sulfate	8 Gm.
Water	50 cc.
2. Sulfurated potash	8 Gm.
Water	50 cc.

Dissolve each separately; filter each, and dispense in two bottles.

Directions: Apply liquid from bottle 2 to affected skin in thin layer, and follow with some from bottle 1.

LOTIONS

Technically, lotions are suspensions of insoluble powders in a fluid. The liquid vehicle adds certain characteristics to the powders.

The evaporation of the fluid vehicle increases the sensation of cooling and causes a further decongestion of the part. The presence of a well chosen fluid medium permits even application. This may be counteracted by unevenness of the sample, as when the powder clings to the container or sinks to the bottom of the bottle. The addition of liquid petrolatum, glycerin or its substitutes may aid in uniform suspension of prescriptions. They do not insure permanence of suspension. Sooner or later the powder ingredients sink to the bottom of the container. The lotion spread on the surface of the skin becomes a false sample of the prescription. Conventional lotions (insoluble powders in liquid) offer ever changing applications to the skin as the insoluble powders remain at the bottom of the jar or bottle and the liquid with the lighter powders is removed.

Bentonite is an ingredient of lotions (insoluble powders in liquid). The bentonite acts to suspend the heavy powders in the fluid medium leading to more elegant-appearing preparations and more equitable samples for each application.

The water-modifying action of sodium hexametaphosphate should be utilized in prescriptions written for the care of the skin and scalp. Water

modified by means of this agent provides softened water for external applications, especially desirable for application of aqueous medicaments on the scalp. Sodium hexametaphosphate for modifying bath water fills a need in areas in which only hard water is available; it permits liberal lathering of conventional soaps and complete removal of any multivalent mineral insoluble soaps formed during the process of washing. The possibility of irritation of the skin through the formation of spicules of calcium or magnesium insoluble soaps within the openings of hair follicles should not be ignored.

Nearly all formularies offer a modification of the lotion through the addition of an oil with free fatty acid and name the result a liniment. The best known is the liniment of calamine and zinc. The vehicle originally was solution of calcium hydroxide, and it is agreed the combination of linseed oil and solution of calcium hydroxide through agitation formed a variety of soap. The method of improving the sample applied to the skin through near neutral soap suspension may be improved by substitution of a modern stearic acid, triethanolamine soap solution, as a vehicle for the suspended insoluble powder ingredients of the lotion-liniment. Calamine, for example, remains suspended in such a modified lotion without settling, and the first application is similar to the last from the one jar.

The following formulas for lotions are suggested:

WATER MODIFIER

Sodium hexametaphosphate	5 Gm.
Distilled water, enough to make	100 cc.

Dilute as required to less than 1 per cent. Use as a vehicle for lotions.

CALAMINE SUSPENSION

Stearic acid	4 Gm.
Triethanolamine	1 Gm.
Water, enough to make	100 Gm.
Zinc oxide	8 Gm.
Prepared calamine	8 Gm.

Melt stearic acid, and mix with triethanolamine dissolved in hot water to form emulsion. Add the powders and stir.

POWDERS

Powders may be defined as solid drugs in finely divided form. The action of the powder applied to the skin depends on certain physical characteristics of the powder and on the surface of the skin. Powder is cooling because it increases the available surface for evaporation of surface fluids. The physical presence of adherent powder affords protection. An absorbent powder acts to dry the surface. Congestion is relieved on the part covered by the powder.

In earlier practice, botanicals predominated in powder base formulas. Flour, starch and orris appeared in the formularies. In modern times, mineral materials are employed. Talc, zinc oxide, kaolin and calcium carbonate are popular. Also, such chemicals as stearates and oleates have displaced many botanicals and minerals.

Certain characteristics of powder formulation must be appreciated before one writes a prescription. There are good characteristics, and there are bad characteristics. Caking, hardening and spiky feel are the bad characteristics. One seeks to avoid them by making the proper choice of ingredients and by proper pharmacy. What are the good characteristics? The powder must be homogeneous. It must spread freely and smoothly. It must have a silky texture. It must adhere to the part. It should give a smooth, soft and pearly finish to the skin.

The degree a powder possesses one or another of the characteristics named depends on the intended site for its application and on the ultimate purpose of its powder nature. A powder designed for the face must have ingredients leading to adherence. A foot powder, for sprinkling into the socks or into the shoes, need not have the character of adherence. A foot powder may be formulated without any adhering materials. Other characteristics of a powder are bulk and coverage.

The dermatologist conscious of pharmacy and pharmacology outlines his prescription for a powder base on this plan: (1) bulk, or volume to weight; (2) slip, or even distribution; (3) coverage, or opaque making, and (4) adherence, or stick. The physician predetermines whether the powder is wholly for one or another of these characteristics; whether it should satisfy more of one than another, or whether there should be a share and share alike proportion.

For a powder to be applied, sprayed or shaken on the skin after a bath, for example, the characteristic sought is bulk, predominantly bulk—much volume for weight. The ingredient selected must have a low density. The carbonates are available. The choice depends on the need for inclusion of other metallic salts, since one ordinarily limits possible conflict by inclusion of one metal ion. Zinc carbonate calls for zinc stearate, for example; magnesium carbonate would be coupled with magnesium stearate. This provision extends only to a few ingredients, not to all. Zinc oxide, for example, is prescribed with other metals, probably because it does not ionize.

A powder may be for the lower portions of the arms or the backs of the hands. Mere bulk does not suffice. The powder must apply evenly; it must have the character of slip. The main ingredient offering slip is the finest grades of talc. Talc is prescribed under the condition named, to satisfy the requirement for slip, for smooth application.

It may be held desirable that the powder possess characteristics of coverage, to cover a blemish, to camouflage the covered surface or to

render the natural skin surface opaque. Two ingredients are available. Zinc oxide gives coverage and is available standardized according to United States Pharmacopeia minimum requirements from pharmacies. Another ingredient giving three times as much coverage for equal bulk as zinc oxide is titanium dioxide. However, the available titanium dioxide has rarely met the severe criteria for low heavy metal content in arsenic set by authorities.

The next character required of many powders is adherence. The powder is designed to stick to the part. Some skin has a self made supply of adherent material in the form of superficial greasiness. Surface discharge of excessive amounts of sweat or sebaceous glands or products of cutaneous irritation acts as adherent quotients. Other skin is poor in such adherent qualities. The stearates supply stick characteristics to a powder. A dry skin requires more stearate than a greasy skin for stick purposes.

The purpose formula given earlier translated into an ingredient formula for a powder base is as follows: zinc carbonate, talc, zinc oxide and zinc stearate. In a mixture of powders it is thought desirable that the metal should be the same for both, as with sodium carbonate and sodium stearate. It is regarded as poor technic to order a mixture of magnesium carbonate and zinc stearate, for example. The prohibition is valid only for certain chemically active powders.

The water-absorbing properties of bentonite, or Denver clay, should be utilized in prescribed powders for contiguous cutaneous surfaces. Foot-dusting powder as well as tooth powder is fortified with advantage by the addition of a small percentage of this clay. Bentonite is an ingredient of modified powder applications, as in the form of a poultice. The dried bentonite resulting from the application of a poultice to the facial skin results in rhythmic self massage and simulates astringent action.

BORATED DUSTING POWDER

Boric acid	4 Gm.
Zinc carbonate	10 Gm.
Zinc stearate	16 Gm.
Kaolin	10 Gm.
Purified infusorial earth	10 Gm.
Talc, enough to make	100 Gm.

DEODORANT FOOT POWDER

Oxyquinoline sulfate	2 Gm.
Zinc oxide	10 Gm.
Talc, enough to make	100 Gm.

SUMMARY

This paper has considered the action and purpose of medicaments prescribed for application to the skin by physicians according to the form

in which medicament is ordered. Bases of creams, greases, solutions, lotions and powders were subjected to study, leading to a rationale of therapy founded on the indicated requirements of the skin to be treated. The choice of the proper base is as important as the inclusion of the active medicament. Empiricism declines in therapeutics so considered. More attention must be given to the phase of response to insult of the skin. It becomes apparent that decongestive lotions by day are not compatible with congestive ointments by night, a device utilized by many physicians and not a few specialists. Official medicaments and drugs must be augmented by patented materials to insure increasingly improved compounding of prescriptions intended for alleviation of cutaneous conditions. The product of research in allied fields, such as the manufacture of synthetics, laundering, food production and animal husbandry, must be utilized. A few products have been named as gathered from these. Many more are already incorporated in professional cosmetics. Illustrative prescriptions are suggested for further experiment. With wider study physicians will devote more attention to prescribing for the patient with a cutaneous disease.

DERMATOPHYTOSIS OF THE HANDS

DIAGNOSIS AND PROGNOSIS

ERVIN EPSTEIN, M.D.
OAKLAND, CALIF.

The concept of dermatophytid has attained universal acceptance. For a long time the extreme swing of the pendulum has almost forced the physician examining a patient with an eruption on the hands and feet to classify the lesions on the hands as a phytid. Therapy based on this reasoning often failed to produce the desired results. In contradistinction, the experience recounted in case 1 in this paper was so dramatic that systematic mycologic examination of all patients with inflammatory lesions of the hands seen in private practice was instituted. Typical hyphae were demonstrated in 5.6 to 7.8 per cent of these patients.

The therapeutic significance of this observation is obvious. It indicates that such lesions of the hands are frequently due to fungi. In cases of this condition it is necessary to apply fungicides locally to obtain the optimum results. Of greater importance is the realization that in 80 to 90 per cent of such cases the eruptions can be classified as one of three distinct types. Furthermore, once the diagnosis is established by appropriate laboratory methods, a definite prognosis can be offered on the basis of the clinical characteristics of the dermatosis. Sabouraud [1] by 1911 had recognized that mycotic infections of the palm differed from those occurring on the dorsal surface of the hand.

The correlation of prognosis with clinical manifestations or with certain strains of organisms was given an impetus by the studies of Lewis and his co-workers on tinea capitis [2] and on infections due to Trichophyton purpureum.[3] This idea has long been recognized in the therapeutic resistance of the achorions. Miliary coccidioidal granuloma presents different cutaneous lesions from those seen in the chronic type.[4]

Read before the Section on Dermatology and Syphilology of the California Medical Association, Del Monte, Calif., May 5, 1941.

1. Sabouraud, R.: Eczematoid Ringworm of the Extremities and Groin, Brit. J. Dermat. **23**:384 (Dec.) 1911.

2. Lewis, G. M., and Miller, H. C.: Ringworm of the Scalp, Arch. Dermat. & Syph. **29**:890 (June) 1934.

3. Lewis, G. M.; Montgomery, R. M., and Hopper, M. E.: Cutaneous Manifestations of Trichophyton Purpureum (Bang), Arch. Dermat. & Syph. **37**:823 (May) 1938.

4. Epstein, E.: Prognostic Significance of Cutaneous Lesions in Coccidioidal Granuloma, Arch. Dermat. & Syph. **38**:752 (Nov.) 1938.

Unfortunately, cultural studies are not presented at this time. However, it may be found that the various eruptions described in this communication are due to specific organisms. It is well established that the third, or hyperkeratotic, form is usually caused by T. purpureum.

FUNGI IN LESIONS OF THE HANDS

Because it seemed that fungi were present more frequently in inflammatory lesions on the hands than is generally considered, a survey of patients encountered in private practice was undertaken. The results are presented in table 1. In each case scrapings from the lesions or shaved vesicle roofs were examined microscopically in a gently warmed solution of potassium hydroxide. Cultures were performed in a few instances.

TABLE 1.—*Inflammatory Dermatoses of the Hands Studied in this Investigation*

Diagnosis	Cases
Dermatophytosis	11
Dermatophytid	14
Eczema	83
Dermatitis venenata	38
Scabies	27
Pyoderma	4
Erythema multiforme	4
Dermatitis medicamentosa	3
Lichen planus	3
Psoriasis	3
Syphilis	2
Pompholyx	2
Disseminated lupus erythematosus	1
Papulonecrotic tuberculid	1

Several of the categories require explanation. In cases in which typical hyphae were demonstrated the condition was called dermatophytosis. Despite the investigations of Dowding and Orr [5] indicating that the mosaic fungus is a form of Trichophyton gypseum, the finding of such bodies was disregarded. Morphologically similar structures were seen in cases of definite dermatitis venenata which cleared with the local application of bland preparations and the removal of the specific irritant.

The pathogenicity of the observed organisms might be questioned in the absence of cultural studies. However, in those instances in which cultures were performed, recognized pathogens were recovered. In addition, the constant observation of a definite clinical picture, the response to therapy and the prognosis in cases in which fungi were found strongly suggest that these organisms were actual causal agents.

5. Dowding, E. S., and Orr, H.: Transformation of Trichophyton Gypseum into Mosaic Fungus, Arch. Dermat. & Syph. **33**:865 (May) 1936.

The cases classified as instances of dermatitis venenata were those in which a specific external irritant was incriminated. A large group of heterogeneous lesions were grouped under the heading of eczema, including unproved dermatitis venenata, housewives' eczema, atopic eczema, orbicular eczema and unclassified dermatoses. The lesions of 2 patients with acute, recurrent, vesicular eruptions apparently precipitated by psychoneurogenous influences were diagnosed as pompholyx.

The incidence of proved dermatophytosis of the hands in this series was 5.6 per cent. If one eliminates the cases of syphilis, pyoderma, psoriasis, lichen planus, poison oak dermatitis, dermatitis medicamentosa, lupus erythematosus, erythema multiforme and scabies—eruptions in which mycotic infection is of less differential diagnostic significance—the incidence rises to 7.8 per cent.

It might be noted that the incidence of reasonably proved dermatophytids was but little in excess of that of the actual mycotic infections of the hands (7.2 to 5.6 per cent). This is in contrast to the opinion expressed by several contributors to a recent symposium.[6] This discrepancy is explained by the fact that these clinicians were probably discussing lifetime observation. The patients were studied during a limited interval in this survey. In many of the patients a dermatophytid of the hands might have developed either prior to or subsequent to this observation. Clinical evidence of pedal dermatophytosis was found in only 54.8 per cent of these cases. In most instances, the interdigital or plantar involvement was mild. It is doubtful if the diagnosis of dermatophytid is tenable in the absence of an active mycotic infection elsewhere on the cutaneous surface or of one affecting its appendages.

REVIEW OF THE LITERATURE

Many problems arise in reviewing the literature. Some of the older statements were based on clinical impression, which is scientifically valueless. Data derived from therapeutic tests is also unreliable. One may fail to cure a T. purpureum infection with fungicides, while a nonmycotic eruption may subside with identical therapy. Many articles fail to state how often organisms were recovered from lesions of the hands. It is also difficult to compare the cases reported in the literature with the ones described here, as the former were often cases in which the investigator was suspicious of a fungous infection before laboratory examination.

---

6. Epstein, E.; Lewis, G. M.; Loveman, A. B.; Pillsbury, D. M.; Schoch, A. G.; Shelmire, B.; Smith, D. C.; Swartz, J. H., and Weider, L. M.: Symposium on the Practical Management of Eczematous Ringworm of the Hands and Feet (Athlete's Foot—Dermatophytosis and Dermatophytids), J. Invest. Dermat. **3**:523 (Dec.) 1940.

However, it is obvious that the finding of fungi in eruptions on the hands cannot be classified as a rarity. Jamieson and McCrae [7] were able to collect a series of 23 cases of such eruptions. Sicoli [8] found fungi in 47 of 113 cases of "dyshidrosis." Lewis, Montgomery and Hopper [3] reported involvement of the hands in 20 of 100 cases of T. purpureum infection. Organisms were recovered from all such lesions. Allington [9] and his associates listed 8 cases of eruptions on the hands in which microscopic examination revealed the presence of mycelium.

Table 2 reviews the reported series [10] lending themselves to statistical compilation. The incidence of positive findings for the entire group of 869 cases is 12.4 per cent. Even if the series of Sicoli and of Darier are eliminated because of the abnormally high findings, the percentage of positive results is still 6.7. It should be remembered that the higher

TABLE 2.—*Review of the Literature* [10] *on the Presence of Fungi in Lesions of the Hands*

Authors	Incidence of Fungi
Lewis, Montgomery and Hopper	in 20 of 100 cases of T. purpureum infections
Sicoli	in 47 of 113 cases of "dyshidrosis"
Tolmach and Traub	in 2 of 32 cases of "epidermophytosis"
Light	in 4 of 179 cases of lesions of the hands
Downing, Merrill and Belding	in 3 of 175 cases of lesions of the hands
Norwood and Evans	in 1 of 20 cases of "glove dermatitis"
Kammer and Callahan	in 2 of 15 cases of "torch oil dermatitis"
Pringle	in 2 of 10 cases of lesions of the hands
Darier	in 11 of 14 cases of "dyshidrosis"
White and Greenwood	in 5 of 15 cases of lesions of the hands
Epstein	in 11 of 196 unselected cases of lesions of the hands (in 11 of 142 selected cases of squamous, vesicular and hyperkeratotic lesions of the hands)

7. Jamieson, R. C., and McCrae, A.: Recurrence or Reinfection in Ringworm of the Hands and Feet, Arch. Dermat. & Syph. **25**:321 (Feb.) 1932.

8. Sicoli, cited by Scholtz, M.: Epidermophytids as a Clinical Conception, Arch. Dermat. & Syph. **25**:812 (May) 1932.

9. Allington, H. V.: Personal communication to the author.

10. (*a*) Tolmach, J. A., and Traub, E. F.: Epidermophytids and the Trichophyton Reaction, Arch. Dermat. & Syph. **28**:560 (Oct.) 1935. (*b*) Light, S. E.: Microscopic Demonstration of Ringworm, ibid. **24**:108 (July) 1931. (*c*) Downing, J. G.; Merrill, B., and Belding, D. L.: A Clinical and Laboratory Study of the Incidence of Fungi in Patients with Cutaneous Eruptions, New England J. Med. **222**:263 (Feb. 15) 1940. (*d*) Norwood, W. D., and Evans, E. E.: Industrial Dermatitis from Gloves, J. A. M. A. **114**:1523 (April 20) 1940. (*e*) Kammer, A. G., and Callahan, R. H.: Torch-Oil Dermatitis, ibid. **109**:1511 (Nov. 6) 1937. (*f*) Pringle, J. J., in discussion on Sabouraud.[1] (*g*) Darier, cited by Scholtz, M.: Epidermophytids as a Clinical Conception, Arch. Dermat. & Syph. **25**:812 (May) 1932. (*h*) White, C. J., and Greenwood, A. M.: Epidermophytosis, J. A. M. A. **77**:1297 (Oct. 22) 1921. (*i*) Lewis, Montgomery and Hopper.[3] Sicoli.[8]

incidence reported by these two investigators may have been due to more diligent study of the cases rather than to any technical error. Perhaps the survey here reported would have disclosed a greater percentage of positive results if there had been opportunity for more extensive investigation of the individual cases. The elimination of the series of T. purpureum infections presented by Lewis, Montgomery and Hopper [3] as constituting a special group still leaves an incidence of 4.7 per cent. All of this indicates that the seemingly high incidence in this series is supported by a review of the literature.

SQUAMOUS TYPE

The squamous type of mycotic infection of the hands is usually easily cured and shows no tendency toward recurrence. It occurs most commonly on the dorsum of one hand. In all cases of this condition in this series men were affected, in contradistinction to cases of orbicular eczema, in which women are more commonly affected. It is characterized by a fairly well marginated, mildly erythematous, scaly or crusted dermatitis. There may be some tendency toward central clearing, but this is often masked. The border is distinct on removal of the desquamation. As a rule the patch is round or oval, but it may be polycyclic. It enlarges slowly by peripheral extension. Although the lesion is most often single, it bears more resemblance to tinea corporis than does either of the other two kinds of dermatophytosis of the hands. The nails are not involved in this form. Chronic interdigital infection of the feet is commonly present.

In the series of cases reported here, this condition has been mistaken for eczema, dermatitis venenata and impetigo.

The following case constitutes a typical instance of this infection.

Case 1.—T. P. B., a 25 year old white man, was seen in July 1938 presenting a dollar-sized pruritic lesion confined to the dorsum of his right hand. The eruption was of two years' duration and during that time he had been examined by a number of general practitioners and dermatologists. All agreed that the eruption could best be classified as eczema. Therapy had included innumerable local remedies, superficial roentgen irradiation and dietary restrictions. He had had numerous scratch tests without the discovery of an etiologic agent. The patient was of the opinion that chocolate caused exacerbations. He admitted intermittent attacks of interdigital "athlete's foot." The nails of his hands were not involved.

A tentative diagnosis of contact dermatitis was made. The patient was exposed to numerous irritants in his work in a vulcanizing shop. However, after three weeks without improvement from measures directed against such a causation and after the performance of thirty-two patch tests with negative reactions, scrapings from the border of the lesion were examined microscopically after gentle warming in a 20 per cent solution of potassium hydroxide. This revealed an abundance of typical hyphae. An ointment containing 1 per cent salicyclic acid and 2 per cent benzoic acid was prescribed for local use twice a day. This

resulted in prompt improvement and a cure within one week. After nearly three years, there has been no recurrence of the condition.

VESICULAR TYPE

Conditions of the vesicular type are comparatively common but are usually classified as dermatophytids of the dyshidrotic type, because of their frequent recurrence and their association with a fungous infection of the feet. However, in 60 per cent of the instances, the lesions on the feet could be described only as mild chronic interdigital maceration, scaling and fissuring. The eruption usually responds rapidly to opening of the blisters and application of fungicidal preparations. Unfortunately, repeated attacks are the rule.

The eruption consists of multiple deep-seated vesicles containing clear fluid. Usually the maximum involvement is on the sides and dorsa of the fingers. Occasionally the entire hand may be affected. Bilateral involvement is more common, although rarely the process may be confined to one hand (case 3). The vesicles may or may not be associated with surrounding erythema. The lesions may dry to form desquamative eczematoid patches. The nails are seldom infected. In all cases in this series comparatively young women were affected. Microscopic examination of the under surfaces of the roofs of the vesicles after immersion in a gently warmed solution of potassium hydroxide usually reveals numerous hyphae.

Some physicians may feel that this condition should be classified as a vesicular phytid. This attitude is questioned because of two observations: In the first place, involvement of the feet accompanying the acute vesicular eruption on the hands is usually inactive, manifesting only a chronic interdigital infection; secondly, organisms have been demonstrated in the lesions of the hands as long as one month after the appearance of an individual patch of lesions. Neither of these facts supports a concept of allergic causation of these lesions.

Case 2 presents a characteristic example of this condition. Case 3 is presented because of two unusual features, i. e., unilateral occurrence and ungual involvement.

CASE 2.—F. P. D., a 15 year old white girl, was seen in November 1939 because of a vesiculobullous eruption involving the sides of all her fingers. There was also one vesicle on the right palm. Although the eruption was of only one week's duration, she had already received diagnoses of dermatitis venenata and scabies. She also presented a mild fungous infection confined to the fourth interspace of each foot. Fungi were demonstrated microscopically from the lesions on the hands. Opening of the vesicles and application of an ointment containing 2 per cent salicylic acid and 4 per cent benzoic acid resulted in complete clearing in ten days. She has had several recurrences since then, all of which have responded to the same therapy.

CASE 3.—Mrs. R. S., a 31 year old white woman, was seen in May 1939 because of a "burning" vesicular eruption which had been present intermittently

on her right hand for five years. She had consulted a number of physicians, both on this continent and in South America. However, she had received little benefit, and no definite diagnosis had ever been made. She presented a few deep-seated vesicles along the lateral surface of her right thumb and on the thenar eminence. She also had a chronic scaling interdigital fungous infection of her feet, which extended to involve her soles. The diagnosis was confirmed as in the previous case. Under the same treatment the lesions cleared in three weeks, but they have recurred on several occasions. The recurrences usually clear within one week with the aforementioned measures. Recently onychomycosis of four of the finger nails of her right hand developed. Her left hand has never been involved.

HYPERKERATOTIC TYPE

The conditions of this kind are reasonably characteristic. They may be confused with palmar and plantar psoriasis, but the involvement of the nail usually furnishes a valuable clue. This type of eruption has also been mistaken for hyperkeratotic eczema or chronic dermatitis venenata.

The lesions are most often confined to the palms but may extend to the sides of the fingers and hands. Occasionally the dorsa are involved. Either one or both hands may be affected. The palms present a dull erythema with cutaneous thickening. Vesiculation is unusual. The patient usually suffers no discomfort, although pruritus may occur. The soles are also affected in most instances. Onychomycosis is the rule. In fact, this is what usually inspires the patient to seek medical aid.

The eruption tends toward extreme chronicity. Five years was the average duration in this series, although in some cases individual lesions had lasted for more than ten years. The usual therapeutic measures, including fractional doses of roentgen rays (75 r) and locally applied fungicides, are comparatively ineffective. In the occasional case in which there seems to be a response, relapses are frequent. This type corresponds to the T. purpureum infections described by Lewis, Montgomery and Hopper.[3] In some instances Epidermophyton inguinale has been recovered from such lesions.[11]

The following case is a typical instance of this condition:

Case 4.—W. N., a 30 year old white man, consulted me in June 1939. He had had an asymptomatic eruption of his left hand and both feet for the past five years. The palm and soles presented a dull erythema with hyperkeratotic scaling. Most of his toe nails and the nails of his left middle and ring fingers were brittle and thickened and lacked luster. Mycologic examination revealed the presence of T. purpureum in the nails and in scrapings from the palm. The diagnosis was confirmed by Lewis after examination of the culture.[12] The patient was presented before the San Francisco Dermatological Society, and the diagnosis was agreed on.[13]

---

11. Lewis, G. M.: Dermatophytosis Due to Epidermophyton Inguinale, Arch. Dermat. & Syph. **40**:623 (Oct.) 1939.

12. Lewis, G. M.: Personal communication to E. A. Levin.

13. Epstein, E.: Trichophyton Purpureum Infection, Arch. Dermat. & Syph. **41**:898 (May) 1940.

The patient was able to control the eruption with an ointment containing 4 per cent salicylic acid and 8 per cent benzoic acid, but slackening in care resulted in a prompt relapse. The infected finger nails and the two great toe nails were removed surgically and have regrown normally. According to the literature, this is an unusual occurrence. This patient has been under the care of various dermatologists during the past two and one-half years.

UNESTABLISHED TYPES

In a small percentage of cases (5 to 10 per cent) insufficient lesions have been observed to allow for definite statements as to morphologic variation and prognostic outlook. The following types seem clinically definite but are presented with the reservations necessitated by the limitation of material.

Desquamative Type.—The following case is an instance of this type of dermatophytosis:

CASE 4.—A 16 year old white girl was observed with desquamation of her finger tips. The involved skin was slightly erythematous but not thickened. The process was of three months' duration and was associated with a severe onychomycosis of nine finger nails and of nine toe nails. Loose pieces of skin were detached and were examined microscopically in a warmed 20 per cent solution of potassium hydroxide. This revealed numerous typical hyphae.

The cutaneous lesions were interpreted as an extension of the ungual infection. While the desquamation cleared rapidly with local fungicidal therapy, the prognosis must be guarded because of the resistance of the basic onychomycosis. Recurrences are to be expected unless the involvement of the nails is eradicated.

Identical cutaneous lesions were seen in a 10 year old boy with one infected nail.

Intertriginous Type.—No instance of interdigital involvement corresponding to the most common of mycotic infections on the feet was noted. Erosio interdigitalis blastomycetica was not included in this study. However, cases of such a condition have been mentioned in the literature, and so this type is included.

Tinea Corporis Type.—Extension of true tinea corporis onto the hands was encountered in only 1 case, in which the causative organism was T. purpureum. Most texts speak of this as a common occurrence.[14] According to the same sources, folliculitis agminata and majocchian granuloma are often noted on the dorsa of the hands. Perhaps a larger series would reveal further unsuspected types. The prognosis of the tinea corporis form should resemble that of the remainder of the eruption

14. Lewis, G. M., and Hopper, M. E.: An Introduction to Medical Mycology, Chicago, The Yearbook Publishers, Inc., 1939. Weidman, F. D., in Blumer, G.: Practitioners Library of Medicine and Surgery, New York, D. Appleton-Century Company, Inc., 1936, vol. 10. Sutton, R. L., and Sutton, R. L., Jr.: Diseases of the Skin, ed. 9, St. Louis, C. V. Mosby Company, 1935.

on the glabrous skin, depending on whether the eruption is caused by an easily destroyed organism, such as Microsporon lanosum, or by a resistant one, such as T. purpureum. In the case of true tinea corporis of the hands previously mentioned, the clinical picture and therapeutic resistance of the hyperkeratotic type were observed.

MIXED TYPES

Unfortunately, the concept as presented is too simple to be all inclusive. Rarely one may find a combination of types. An illustrative case is that of a man with a vesicular mycotic eruption of his feet and a vesicular dermatophytid on his hands. When seen one year later, he presented a hyperkeratotic form of fungous infection on his hands and both vesicular and hyperkeratotic lesions on his feet.

The hands are intimately concerned with the environment. This plus ill advised medication may superimpose a dermatitis venenata on any of the described types of infection. In such instances recurrences may be due to environmental factors rather than to the fungi themselves. The finding of an organism does not eliminate the possibility of an associated contact dermatitis.

Also, there is no reason why a fungous infection could not develop in an area of dermatitis due to a noninfectious factor. This, too, would alter the clinical appearance of the mycotic infection. These situations would constitute exceptions to the previously described fungous eruptions of the hands.

As this survey is based on only about 75 cases of fungous infections of the hands, which were observed personally, studied through the courtesy of other physicians or reported completely enough in the literature to be of value, my statements cannot be considered as unalterable facts. The limitation of material is such that the concepts expressed here must be looked on merely as indications pointing to the need for further study of this most important subject.

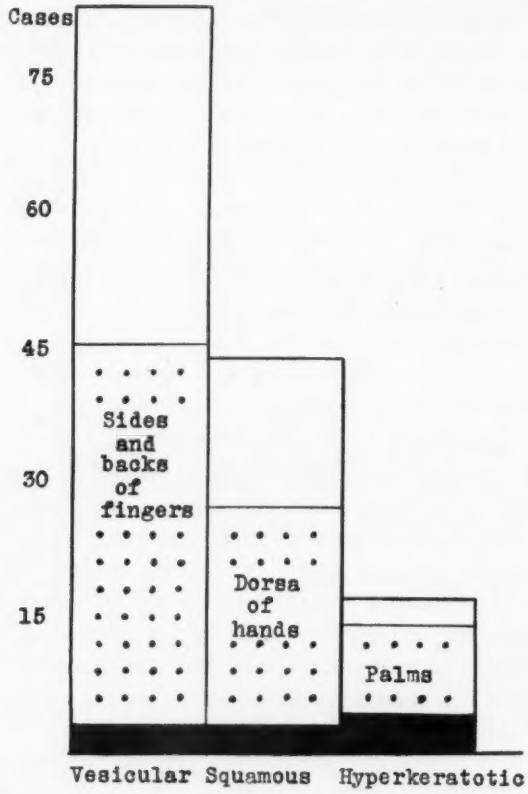
COMMENT

One often hears the statement that fungous infections of the hands can be diagnosed by their clinical appearance. While it is true that certain clinical manifestations are seldom caused by mycoses, the mixed type of fungous infection with an associated dermatitis venenata may be indistinguishable from any vesicular, squamous or eczematoid eruption occurring in this location. Furthermore, any of the dermatophytoses I have described may be mimicked by nonmycotic lesions.

These impressions were gained by the analysis of a series of 142 cases of hyperkeratotic, squamous and vesicular lesions of the hands. The vesicular eruptions due to poison oak and scabies were not included.

Lesions elsewhere on the cutaneous surface usually make the diagnosis obvious without recourse to mycologic investigation.

Many of the observations of this study are shown in the accompanying chart. According to the chart, vesicular lesions on the hands are more common than those described as squamous or hyperkeratotic. Also, none of the basic types of lesions is clinically pathognomonic of a fungous infection. Fungi were found in only 3.7 per cent of the vesicular



Observations made in 142 cases of vesicular, squamous and hyperkeratotic eruptions occurring on the hands. The filled-in spaces indicate cases of dermatophytosis; the spaces containing dots indicate cases of eruptions occurring in characteristic locations; the clear spaces indicate cases not included in other groups.

eruptions, 7.5 per cent of the squamous dermatoses and 29.4 per cent of the hyperkeratotic lesions on the hands. In other words, all three are more commonly due to other agents.

The chart also indicates that one is most apt to find fungi in hyperkeratotic lesions and least likely to recover them from vesicular eruptions.

It also suggests that the location of the eruption is not of great diagnostic significance. Most of the squamous dermatophytoses occur on the dorsum of the hand. Yet 64.4 per cent of all squamous eruptions occurred on this surface, and in only 11.1 per cent of these were fungi found. The hyperkeratotic type usually involves the palms, but 76.6 per cent of all hyperkeratotic lesions involved this area. Fungi were demonstrated in the high percentage of 38.5 per cent of the palmar hyperkeratotic dermatoses. Sixty-one and seven-tenths per cent of the dyshidrotic lesions involved the sides and dorsa of the fingers and hands, the most common location for the vesicular type. Fungi were seen in only 4.8 per cent of the cases of this type.

Some claim that the presence or absence of dermatophytosis of the feet is of importance in diagnosing lesions of the hands. As stated

TABLE 3.—*Differentiation of Three Major Types of Dermatomycotic Infection of the Hands*

	Type 1: Squamous	Type 2: Vesicular	Type 3: Hyperkeratotic
Clinical characteristic	Superficial	Deep-seated	Thickened
Location	Dorsum of hand	Sides of fingers	Palms
Involvement	Unilateral	Bilateral	Either
Pruritus	Usually	Seldom	None
Duration	Weeks to months	Weeks to years	Years
Onychomycosis	None	Usually none	Common
Similar involvement of feet	None	Variable	Common
Sex (most commonly)	Male	Female	Male
May be confused with	Eczema, impetigo	Dermatophytid, pompholyx	Psoriasis, eczema
Response to therapy	Excellent (4 to 10 days)	Good (10 to 30 days)	Poor (months to years, if at all)
Recurrences	None	Usually	Usually

previously, 54.8 per cent of the patients showed such a pedal infection, while dermatomycosis of the hands was seen in but 7.8 per cent of the instances. Involvement of the foot was noted in 54 per cent of the cases of all vesicular eruptions, in 43.4 per cent of the cases of squamous eruptions and in 88.8 per cent of the cases of hyperkeratotic lesions. However, in the patients with proved dermatophytosis, the feet were affected in 100 per cent of those with the vesicular and hyperkeratotic forms but in only 33.3 per cent of those with the squamous lesions. To summarize: A fungous eruption of the hands is usually accompanied by involvement of the feet. The converse does not hold true, as one sees many examples of infection of the feet unaccompanied by similar lesions of the hands.

Studies of the duration of the lesions when seen were unproductive. All types of lesions may persist for years. Their duration depends on the length of time necessary for the patient to receive an accurate diag-

nosis and adequate therapy. The duration of the vesicular and squamous dermatoses varied from weeks to years. The hyperkeratotic lesions were all of long duration. This is partially explained by the paucity of symptoms. The hypertrophy of the corneous material also renders proprietary and home remedies ineffective.

Combinations of clinical characteristics did not prove any more typical than the aforementioned factors. It cannot be denied that experience leads to clinical acumen that cannot be attributed to pathognomonic features capable of description. Yet, such factors as grouping and central clearing did not prove of great value in the recognition of the conditions. Wise and Wolf [15] have pointed out that the diagnosis of dermatophytid requires careful consideration of many factors rather than simple inspection of the hands and feet. The same holds true for actual mycotic infections of the hands. In conclusion, recourse to the microscope and culture tube constitutes the most accurate method of diagnosis in cases of dermatophytosis of the hands.

SUMMARY

In 5.6 per cent of 196 unselected patients with inflammatory lesions of the hands, typical mycelium was demonstrated in the lesions.

In 7.8 per cent of 142 selected patients fungi were found. The eruptions of these patients could be divided into several groups, as follows:

1. The squamous type cleared easily and showed no tendency toward recurrence.

2. The vesicular type responded rapidly but tended to recur.

3. The hyperkeratotic type was resistant to therapy.

4. Mixed and unclassified types also occurred but were in the minority.

The diagnosis of dermatophytosis of the hands must rest on laboratory investigation.

15. Wise, F., and Wolf, J.: Dermatophytosis and Dermatophytids, Arch. Dermat. & Syph. **34**:1 (July) 1936.

DEPROTEINATED PANCREATIC EXTRACT IN TREATMENT OF PSORIASIS

JOHN G. DOWNING, M.D.

EARL A. GLICKLICH, M.D.

AND

SALVATORE J. MESSINA, M.D.

BOSTON

The disturbance of fat metabolism as a causative agent or a contributing factor in psoriasis has been the subject of many investigations. Recent work has shown that an alcoholic extraction of raw pancreas from which lipid materials have been excluded by ether extraction, i. e. lipocaic, appears to play some role in the metabolism or transport of fat. When given to depancreatized dogs who are receiving insulin and are on a normal diet, it prevents the fatty infiltration which usually develops in these animals. It has been found to reduce the abnormally high lipid content of the blood of persons with xanthoma.[1]

There is no complete agreement that lipocaic is a hormone. Dragstedt has persisted in considering this noncholine lipotropic component of pancreas to be a hormone.[2] This view has been vigorously attacked by Chaikoff in a series of papers in the *Journal of Biological Chemistry*. Whereas Dragstedt has maintained that the external secretion of the pancreas is incapable of supporting normal lipid metabolism in the liver, Chaikoff and others appear to have almost incontrovertible evidence that pancreatic juice contains large amounts of a "lipocaic-like" substance. This appears to indicate that the pancreas secretes the substance into the pancreatic juice for use within the lumen of the intestine and renders Dragstedt's claim for hormonal activity of doubtful validity. It should be emphasized, however, that, whether it is a hormone or not, there is

1. Stewart, C. D.; Clark, D. E.; Dragstedt, L. R., and Becker, S. W.: The Experimental Use of Lipocaic in the Treatment of Psoriasis, J. Invest. Dermat. **2**:219-230, 1939. Walsh, E. N.; Clark, D. E.; Dragstedt, L. R., and Becker, S. W.: Further Observations on the Use of Lipocaic in the Treatment of Psoriasis, ibid. **4**:59-67, 1941.

2. Dragstedt, L. R.; Van Prohaska, J., and Harms, H. P.: Observations on a Substance in Pancreas (a Fat Metabolizing Hormone) Which Permits Survival and Prevents Liver Changes in Depancreatized Dogs, Am. J. Physiol. **117**:175-181, 1936.

a substance in the pancreas of great potency in preventing the development of fatty livers in depancreatized dogs.[3]

One group of investigators reported two studies [1] on the experimental use of lipocaic in the treatment of psoriasis. It was first given to 6 patients; 4 showed decided improvement, and 2 failed to show any response. In this study it was thought that lipocaic lowered the lipid content of blood serum to normal or below normal level and that improvement depended on maintaining the serum lipids at the lower limits of normal or below. In the second study 23 patients were observed for from two to eighteen months. After the oral administration of lipocaic alone, clearing of the lesions occurred in 31 per cent, improvement in 50 per cent and no change in 19 per cent. Lipocaic did not

Results of the Use of Deproteinated Pancreatic Extract in the Treatment of Psoriasis

Patient	Duration of Disease	No. of Injections	Amount in Cc.	Weeks of Observation	Results
G. P.	20 yr. intermittently	20	54	4	Complete regression of lesions
D. K.	4 yr. intermittently	60	178	12	Decided improvement
M. G.	20 yr. intermittently	57	165	14	Decided improvement
D. A.	7 yr. intermittently	26	77	20	Decided improvement
M. Mc.	2 yr. intermittently	51	153	10	Slight improvement
V. W.	1 yr.	28	83	8	Slight improvement
H. O.	3 months	16	48	12	Slight improvement
E. O.	8 months	62	182	14	No improvement
G. R.	3 yr. intermittently	12	36	4	No improvement
B. F.	10 yr. intermittently	61	180	12	No improvement
J. Mc.	8 months	50	149	16	No improvement
S. M.	6 yr. intermittently	47	141	12	No improvement
C. D.	4 yr. intermittently	9	27	4	No improvement

cause a reduction of the lipid level of the blood of patients with psoriasis, as had been thought, but the lesions of a patient with a low lipid content of the blood cleared more readily than those in a patient with a high lipid level of the blood.

In view of these reports it was deemed worth while to try another type of pancreatic preparation, a deproteinated extract, in the treatment of psoriasis. The extract used [4] is a colorless saline solution of a chemically purified, protein-free, nitrogenous fraction, derived from an acid-alcohol extract of beef pancreas. Physiologic tests show that it is free of insulin, histamine and acetylcholine. It contains approximately 2.5 per cent of solids, including 0.25 per cent of nonprotein nitrogen, 0.9 per cent of sodium chloride and 0.25 per cent of phenol as a preservative. It is adjusted to a p_H of 6.5 to 6.8.

3. Henderson, J.: Personal communication to the author.

4. This extract, depropanex, was supplied by Sharp & Dohme.

Our work was confined to observing the clinical effects of this drug, and no lipid studies were performed. Thirteen patients were given intramuscular injections of the substance, the dose ranging from 1 to 3 cc. daily, every other day or once a week. Throughout the course of therapy no toxic symptoms were observed. In only 1 patient did the condition clear completely; 3 showed decided improvement; 3 slight improvement, and 6 no improvement.

The results obtained from the use of the deproteinized pancreatic extract in the treatment of psoriasis, while disappointing, were what we expected. Almost any new therapeutic agent, especially when given by injection, seems to have a beneficial effect on a small group of psoriatic patients. It was interesting to note that all who were benefited were females. Nevertheless, in view of the favorable reports on lipocaic and of the beneficial effect, in 50 per cent of these patients, further studies in the use of pancreatic extracts in the treatment of psoriasis should be encouraged.

520 Commonwealth Avenue, Boston.

Fort Devens, Mass.

520 Commonwealth Avenue, Boston

HEMOCHROMATOSIS

REPORT OF A CASE AND COMMENT ON METHODS OF MAKING A DEFINITE DIAGNOSIS

ARTHUR A. HUMPHREY, M.D.

SAM ALPINER, M.D.

AND

L. E. VERITY, M.D.

BATTLE CREEK, MICH.

Since the literature now records almost 400 acceptable cases of hemochromatosis, the value of adding further cases is dubious unless they facilitate the clinical diagnosis of the disease. Despite the use of the cutaneous biopsy and the urinary hemosiderin test of Rous,[1] clinical diagnosis still leans heavily on the classic triad of pigmentation, diabetes mellitus and cirrhosis of the liver. Since one or two of the three may be lacking, the bulk of the diagnoses still are made in the necropsy room. Butt and Wilder [2] reviewed 32 cases in 1938 and failed to make a diagnosis in but 2 of the cases; on the contrary, Mills [3] in 1934 reported a diagnosis during life in but 2 of 17 cases, and his experience appears to be that generally encountered.

Recently, in cases of hemochromatosis with diabetes mellitus, treatment of the diabetes has proved to be of definite value, and patients have lived for as long as thirteen years after the original diagnosis was made. If the recent work on its causation which has been performed from the avitaminosis standpoint bears fruit, there may be a solution for other manifestations of this disease.

In the following case diabetes was absent and the pigmentation displayed a confusing intermittency. The iron pigment characteristic of the disease, hemosiderin, was demonstrated in the urine, in the ascitic fluid and in a biopsy specimen of the skin.

REPORT OF CASE

History.—F. D., a Polish man aged 41, a motor valve grinder, was admitted to Leila Y. Post Montgomery Hospital on April 21, 1939 complaining of extreme

From the Pathological Laboratory, Leila Y. Post Montgomery Hospital.

1. Rous, P.: Urinary Siderosis, J. Exper. Med. **28**:645-658 (Nov.) 1918.

2. Butt, H. R., and Wilder, R. M.: Hemochromatosis, Arch. Path. **26**:262-273 (July) 1938.

3. Mills, E. S.: Hemochromatosis with Special Reference to Its Frequency and to Its Occurrence in Women, Arch. Int. Med. **34**:292-300 (Sept.) 1924.

weakness, discoloration of the skin and swelling of the abdomen and lower extremities. There was no history of diabetes or of unusual pigmentation of the skin in the family. In 1933 he first noticed a brown pigmentation of the skin. His general health was good until a year previously, at which time he complained of dyspnea and swelling of the feet and legs. These conditions at first would disappear on resting, but in the last six months they persisted and in the last two weeks became much more pronounced. In January 1939 ascites was first noticed. None of the usual treatments for this condition produced any decided diuresis. Dyspnea became more troublesome, and on admission there was considerable cyanosis of the lips and nails. At no time did he give a history of diabetes or complain of polyuria or unusual thirst.

His past history was irrelevant except for occasional indulgence in alcohol and exposure to metals.

Physical Examination.—Physical examination disclosed a peculiar dusky gray-brown discoloration and atrophy of the entire skin. The mucous membranes of the throat and buccal cavity appeared dusky. The veins of the neck were prominent. The pulmonary fields were clear throughout except for some bronchovesicular breathing at the bases, and the heart sounds were weak and distant. His blood pressure was 90 mm. of mercury systolic and 62 mm. diastolic; his pulse rate was 110 beats per minute and his temperature 98.8 F.

Considerable free fluid was present in the abdomen. The upper border of liver dulness was at the fourth rib and the lower edge was palpable to the umbilicus in the midclavicular line. The edge was sharp, smooth, firm and not tender. The spleen was barely palpable. There was a 3 plus pitting edema of the lower limbs and thighs.

Laboratory Studies.—On entry, the erythrocytes numbered 4,640,000 per cubic millimeter; the hemoglobin amounted to 70 per cent; the leukocytes numbered 8,950 per cubic millimeter, and the differential count was essentially normal. The Kahn reaction was negative. A fasting blood sugar value was 100 mg. per hundred cubic centimeters. Examination of the stool revealed some undigested food but no lack of pancreatic ferments.

The urinalysis showed no abnormalities with the exception of many hemosiderin granules in the sediment, which were identified by the use of 2 per cent hydrochloric acid and 2 per cent potassium ferrocyanide under a cover glass.

Course.—On April 22, 1939 15 cc. of clear yellow ascitic fluid was withdrawn and examined for tumor cells. Hemosiderin granules were observed as golden brown granules in the macrophages and identified by the prussian blue reaction.

The following day some reduction in pigmentation of the skin was noted, which persisted for several days before the original darker hue returned. A specimen of skin for biopsy was taken on April 24, and hemosiderin granules were demonstrated in fresh frozen and fixed sections.

In spite of a high carbohydrate diet and intravenous dextrose medication the patient became progressively weaker and died one week after admission.

Autopsy.—The body was well developed and not emaciated. The skin was grayish brown with no variation in shade throughout its surface. The subcutaneous tissue was so edematous that water exuded when it was cut.

The heart was slightly enlarged, and on cross section the myocardium was reddish brown. The pericardial sac contained 35 cc. of clear fluid. There was a grade 1 sclerosis of the aorta and the coronary arteries.

The left pleural cavity contained 600 cc. of slightly cloudy straw-colored fluid. The lung was free in this fluid, and there were an increase in consistency in the lower lobe and on section a lack of froth and an increase in fluid in this area. The right lung was adherent to the parietal pleura by firm adhesions and on section was similar to the left.

The gastric mucosa was faintly nodular and was dusky red; the remainder of the gastrointestinal tract was normal, but the colon also possessed this same rusty hue.

The spleen weighed 500 Gm. and was dark red; on cross section it appeared firm and fibrous. The liver was increased 40 per cent in size (2,200 Gm.); the surface was nodular, and it was reddish brown. It was sectioned with difficulty, the knife progressing slowly as if cutting thick leather. On cross section it was rust brown and the markings were not increased in prominence. The gallbladder and the bile ducts appeared normal.

The pancreas was red-brown, and the fat replacement was decided. The adrenals appeared normal. The kidneys were slightly enlarged, and on section there was an increase in the amount of peripelvic fat.

The diagnosis after gross examination was hemochromatosis.

Microscopic sections through the liver showed a great increase in fibrous tissue. There was a splitting of the lobules into definite islands, which were surrounded by fibrous tissue bands. Typical cirrhosis was present. Throughout the entire liver there were deposits of brown hemosiderin pigment in the fibrous tissue and a diffuse phanerosis of hemosiderin in the liver cells themselves. It was also present in the heart muscle in certain areas. There were masses of pigment in the pancreas. The fat replacement in the pancreas was considerable, and the islands appeared to be numerous. Sections through the lung tissue demonstrated a few cells filled with hemosiderin pigment.

COMMENT

Clinical descriptions of the disease are so numerous and complete that we can restrict our discussion to the definite diagnosis of the disease, i. e., by the microchemical detection of hemosiderin in the body fluids and tissue. It is worthy of note, however, that this patient did not have glycosuria or hyperglycemia and that his pigmentation, which was gray-brown, actually changed in intensity while he was in the hospital, becoming definitely lighter and then darkening before death. This variation has been noted by a number of authors in several patients under insulin therapy, and Lawrence [4] attributed it to a varying volume of water and glycogen in the superficial cells of the skin, which lie external to the pigmented portions. Thus, in our case the pigmentation probably decreased as the general tissue edema increased.

Rous,[1] in 1918, reported a method for demonstrating hemosiderin in the urine by the use of the prussian blue reaction. He mixed fresh human serum with the centrifuged sediment and made films, which he treated with 2 per cent potassium ferricyanide and 1 per cent hydro-

4. Lawrence, R. D., cited by Sheldon, J. H.: Haemochromatosis, London, Oxford University Press, 1935, p. 34.

chloric acid in equal parts; he used 1 per cent hydrochloric acid alcohol for further contrast and dehydrated and placed them under cover slips, sealing with balsam, for permanent mounts. He found no iron pigment in 33 control cases but found it present in 8 of 10 cases of pernicious anemia, although not in the tissue cells. It is conceivable that in certain blood dyscrasias a positive reaction of this sort should be weighed somewhat carefully. Butt and Wilder [2] found urinary hemosiderin in but 2 of 7 cases and did not mention the method. In the present report the urinary sediment was mixed directly with the two reagents and viewed while fresh under a cover slip. The granules were large, bright blue and numerous. The cases in which this test can be done certainly warrant its use before a biopsy is attempted.

Ascites is present in the bulk of cases at necropsy, but in the earlier stages, in only a third of the instances or less. When it is removed, however, for symptomatic relief or to search for tumor cells, as in the present instance, the ferricyanide and hydrochloric acid treatment of the sediment may demonstrate the hemosiderin-filled cells and strongly suggest the diagnosis of hemochromatosis. It must be borne in mind that the phagocytosed old blood pigment of intra-abdominal hemorrhages might also present the same picture and should be ruled out. We are not aware of the previous use of this diagnostic procedure, but the photomicrograph of the same sediment on opposite ends of the same plate demonstrates the striking contrast between the unstained portion and that which has reacted with the ferrocyanide.

In but a few instances has biopsy of the skin failed to reveal hemosiderin in these cases. The skin can be sectioned and stained with any of the ferrocyanide or ferricyanide methods or by the use of dinitroresorcinol; by employing only ammonium sulfide, one may fail to distinguish the pigment from that observed in argyria. In the present case it was even demonstrated in frozen sections by means of the free reagents, and the pigment appeared to be greater about the propria of the sweat glands and in a lesser amount in the stratum malpighii.

Rous mentioned the discovery of a laden granulocyte in the blood stream in which he demonstrated the pigment with the Nishimura reaction. This feat was the result of a laborious search, and one might reflect that the possibility of an occasional *Herzfehlerzelle* in the peripheral circulation is far from impossible.

Fishback [5] has recently described an intradermal test which may prove of value: Equal parts of sterile potassium ferrocyanide and hundredth-normal hydrochloric acid promptly produce a blue wheal when injected into the skin. The ease with which small metallic particles on the micro-

5. Fishback, H. R.: Clinical Demonstration of Iron in the Skin in Hemochromatosis, J. Lab. & Clin. Med. **25**:98-99 (Oct.) 1939.

tome knives give the characteristic color reaction in sections should make thoughtful users first rule out rust and such particles as are produced by grinding the needle tip.

SUMMARY

A case of hemochromatosis is reported in which diabetes was not present and the pigmentation displayed an unusual variation in intensity.

Our complete lack of knowledge concerning the cause of this disease has interfered with successful treatment. It would appear, however, that adequate control of the diabetes has been a most important factor in prolonging the lives of these patients.

When ascitic fluid is available, as in the present instance, it is suggested that the microchemical reactions for iron pigments be performed in its sediment as a routine diagnostic aid in hemochromatosis.

While a biopsy of skin is of the greatest value in the diagnosis of this disease, the simpler urine test for hemosiderin should be attempted first, the clinician being cognizant of its limitations.

Leila Y. Post Montgomery Hospital.

GEOGRAPHIC DISTRIBUTION OF LUPUS ERYTHEMATOSUS

EMANUEL GAHAN, M.D.
NEW YORK

There is a general belief that actinic trauma is a factor in the causation or pathogenesis of lupus erythematosus. I have examined the literature in order to determine whether any correlation exists between the relative frequency of this disease and climatic conditions prevailing throughout the world. Adequate statistics based on large numbers are not always obtainable; it is often necessary to rely on the opinions of authorities based on personal observations over varying periods.

In the dermatologic clinics of the United States approximately 1 out of every 200 to 400 new patients has lupus erythematosus. The disease is, as MacLeod [1] called it, one of the more common of the rarer dermatoses. Pollitzer [2] gave the incidence as roughly 1 in 300. There is nothing in the voluminous literature on the disease to indicate that it is any more common in one part of the country than in any other part (table 1).[3]

In Cuba the climate resembles that of southern Florida. "It is surprising," said Pardo-Castello,[4] "to find that lupus erythematosus is no more frequent in Cuba than in the United States."

For South America reliable first hand information comes from an excellent memoir by Rabello,[5] of Rio de Janeiro, Brazil, who gave not only his own statistics over a period of years but also those of other dermatologists in Brazil, Uruguay and Argentina (table 2). The territory covered by this study and the variations in climate are as great as those of the United States. Rio de Janeiro has warm, humid weather with an average temperature of 74 F., varying little from month to month; São Paulo, the second city in Brazil, has a more temperate

1. MacLeod, J. M. H.: Lupus Erythematosus, Arch. Dermat. & Syph. **9**:1 (Jan.) 1924.

2. Pollitzer, S., in discussion on MacLeod.[1]

3. (*a*) Cummer, C. L.: Etiology of Lupus Erythematosus, Arch. Dermat. & Syph. **33**:434 (March) 1936. (*b*) Weiss, R. S., in discussion on Cummer.[3a] (*c*) Lane, C. G., in discussion on Cummer.[3a] (*d*) Gahan, E.: Lupus Erythematosus: Clinical Observations in Four Hundred and Forty-Three Cases, Arch. Dermat. & Syph. **45**:685 (April) 1942. (*e*) American Dermatological Association figures, cited by R. Spitzer (in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1928, vol. 14, pt. 2, p. 317).

4. Pardo-Castello, V., in discussion on Cummer.[3a]

5. Rabello, E.: La tuberculose cutanée; son incidence dans quelques régions de l'Amérique du Sud, Eighth Internat. Cong. Dermat., Copenhagen, 1930, p. 381.

climate and is located at an altitude of 2,500 feet (762 meters) above sea level; Montevideo, Uruguay, and Buenos Aires, Argentina, have weather which resembles that of Norfolk, Va. It appears from these statistics, gathered by competent observers over many years, that lupus erythematosus is at least as common in these localities as it is in New York.

In England, where the climate is wet and mild, the disease is common, as is shown by the statistics in table 3,[6] which are the only ones available.

TABLE 1.—*Incidence of Lupus Erythematosus in the United States*

City	Years	Total New Cases of Cutaneous Diseases	Lupus Erythematosus	
			Cases	Per Cent
St. Louis [5b]	1905-1935	34,384	220	0.64
Boston [5c]	1930-1935	22,000	85	0.39
New York [5d]	1936-1940	153,475	443	0.29
Various cities [5e]	1887-1910	679,376	2,579	0.38

TABLE 2.—*Incidence of Lupus Erythematosus in South America*

City	Author	Total New Cases of Cutaneous Diseases	Lupus Erythematosus	
			Cases	Per Cent
Rio de Janeiro	Rabello	4,972	19	0.38
São Paulo	Pupo	21,516	54	0.25
Montevideo	Foresti	20,000	145	0.725
Buenos Aires	Balina	12,553	103	0.82

TABLE 3.—*Incidence of Lupus Erythematosus in England*

City	Author	Total New Cases of Cutaneous Diseases	Lupus Erythematosus	
			Cases	Per Cent
London	MacLeod [1]	20,000	300	1.5
London	Crocker [6b]	10,000	63	0.63
Manchester	Wild [6c]	30,000	142	0.47

For France the only figures I have been able to find are those of Dubreuilh,[7] of Bordeaux, compiled at least forty years ago; he gave an incidence of 1.1 per cent. The disease is probably common, if one may judge from the large number of papers devoted to it in the French literature. At a special meeting of French-speaking dermatologists, held at

6. (*a*) MacLeod.[1] (*b*) Crocker, H. R.: Diseases of the Skin, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1903, p. 1402. (*c*) Wild, R. B., in discussion on MacLeod, J. M. H.: Discussion on the Nature, Varieties, Causes and Treatment of Lupus Erythematosus, Brit. M. J. **2**:316, 1913.

7. Dubreuilh, cited by Voirol.[8]

Strasbourg, France, in 1939, the sole topic of discussion was lupus erythematosus.

For central Europe, Voirol,[8] in 1903, gave the information contained in table 4.

In recent years the statistics[9] given in table 5 have been made available.

In his article on lupus erythematosus in Zagreb, Yugoslavia, Kogoj [9e] made these significant remarks:

One finds in almost all dermatologic textbooks the statement that lupus erythematosus is a disease which is rare in southern, sunny countries. Yugoslavia

TABLE 4.—*Incidence of Lupus Erythematosus in Central Europe in 1903*

City	Incidence
Berlin, Germany	0.15
Canstatt, Germany (Veiel)	1.40
Halle, Germany (Kromeyer)	0.46
Hamburg, Germany (Unna)	0.11
Königsburg, Germany	0.90
Munich, Germany	0.97
Nuremberg, Germany	0.57
Zurich, Switzerland	0.33
Berne, Switzerland (Jadassohn)	1.28

TABLE 5.—*Incidence of Lupus Erythematosus in Central Europe*

City	Years	Lupus Erythematosus	
		Cases	Per Cent
Kiel, Germany [9a]	1911-1913, 1920-1924	112	0.59
Breslau, Germany [9b]	1911-1913, 1919-1925	251	0.71
Zagreb, Yugoslavia [9c]	1928-1939	385	0.85
Zurich, Switzerland [9d]	1917-1938	206	0.206
Budapest, Hungary [9e]	1893-1914	Not given	0.60

is not a northern country, and yet cases of lupus erythematosus comprise almost 1 per cent of all our cases; besides, out of 385 cases there were 42 (one-ninth)

8. Voirol, A.: Ein Beitrag zur Aetiologie und Kasuistik des Lupus erythematodes, Deutsche Med.-Ztg. **24**:885, 1903.

9. (*a*) Krusewitz: Statistik der Hautkrankheiten der Kieler Univ. Hautklinik, Inaug. Dissert., Kiel, 1925; cited by Spitzer, R., in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1928, vol. 14, pt. 2, p. 314. (*b*) Hernig, H.: Statistik der Hautkrankheiten der Breslauer Univ. Hautklinik, Inaug. Dissert., Breslau, 1927. (*c*) Kogoj, F.: Quelques données sur les cas de lupus érythémateux observés dans la clinique dermato-vénéréologique de Zagreb, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **46**: 1064, 1939. (*d*) Miescher, G.: Résultats statistiques sur les rapports entre le lupus érythémateux et la tuberculose, ibid. **46**:1056, 1939. (*e*) Somogyi, S.: Der heutige Stand der Frage des Lupus erythematodes, Zentralbl. f. Haut- u. Geschlechtskr. **18**:641, 1925.

from coastal regions (many of which were hundreds of kilometers from Zagreb), where it is sunny.

In southern Europe the disease is probably no rarer than it is in central Europe. Covisa,[10] of Madrid, Spain, reported on 309 personally studied cases; he gave no percentages, but this was a larger number of cases than most of the other observers had been able to collect. On the Italian Riviera, Truffi,[11] of Padua, stated that the disease was one of the most frequently seen of all dermatoses. For the Adriatic coast there was the report of Kogoj.[9c] In Greece the disease was widespread, according to Kelemenis,[12] of Athens.

In Angora, Turkey, where the summers are warm and dry and the winters are cold, Marchionini [13] encountered 8,736 new cases of cutaneous diseases in his clinic in 1938, of which 19 were of lupus erythematosus (0.22 per cent). The same author reported that the disease was seen more often in the clinics of the large cities along the Black Sea and the Mediterranean coast, where the summer temperature is like that of the central plateau on which Angora is located but where the humidity is higher.

In Sydney, Australia, where the climate is warm and sunny (there are more than two thousand hours of sunshine per year), Molesworth [14] stated that the disease was seen approximately as often as it was in the clinics of Europe. Cancer of the skin, on the other hand, was seen far more often.

In Japan, Uchido [15] reported the incidence of lupus erythematosus as 0.23 per cent. Recently Ito and Yamanouti [16] discussed its causation apropos of 7 cases, without mentioning anything about dearth of material for the study. In Hasselmann's article [17] on the disease in the Philippine Islands he reported 5 cases, of which 3 were of Japanese patients.

---

10. Covisa, J.: La tuberculose cutanée, Eighth Internat. Cong. Dermat., Copenhagen, 1930, p. 365.

11. Truffi, M.: Lupus eritematoso in tre sorelle, Gior. ital. d. mal. ven. **65**:723, 1924; abstracted, Zentralbl. f. Haut- u. Geschlechtskr. **14**:85, 1924.

12. Kelemenis, B.: Das Gold in der Therapie des Lupus erythematodes, Athen. kliniki, 1926, p. 286; abstracted, Zentralbl. f. Haut- u. Geschlechtskr. **20**:804, 1926.

13. Marchionini, A.: Observations climatologiques en Anatolie Centrale en rapport avec la pathogénie du lupus érythémateux, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **46**:1030, 1939.

14. Molesworth, E. H.: Is Lupus Erythematosus a Tuberculide? Urol. & Cutan. Rev. **37**:62 (Sept.) 1933; Introduction to Dermatology, London, J. & A. Churchill, 1937.

15. Uchido, S.: On Psoriasis Vulgaris and Lupus Erythematodes, Jap. J. Dermat. & Urol. **27**:14, 1927.

16. Ito, M., and Yamanouti, G.: Lupus Erythematodes: Etiology and Pathogenesis, Jap. J. Dermat. & Urol. **47**:36, 1940.

17. Hasselmann, C. M.: Lupus Erythematosus (Discoideus) in Tropics, Arch. Dermat. & Syph. **29**:585 (April) 1934.

In tropical countries inhabited by colored races the disease is apparently extremely rare. Noel [18] reported, in 1928, the first case found among the inhabitants of southern India. Hasselmann in 1935 reported the first example of lupus erythematosus in a Filipino.

CONCLUSIONS

If 0.40 per cent is taken as the incidence of lupus erythematosus in the United States, there is no appreciable difference between the relative frequency of the disease here and in any other part of the world inhabited by the white race; almost nowhere is it less than half as common or more than twice as common. At any rate, whatever differences there may be are not based on climatic conditions. The frequency of the disease is as unpredictable and capricious as its evolution in patients.

12 West Forty-Fourth Street.

18. Noel, P.: Le lupus érythémateux dans les races noires, Ann. de dermat. et syph. **9**:372. 1928.

HISTORY OF THE CHICAGO DERMATOLOGICAL SOCIETY

MAX S. WIEN, M.D.
CHICAGO

At one of the annual meetings of this society Dr. Otto H. Foerster [1] made the following remarks:

> A spirit of mutual regard and of good fellowship seems to be characteristic of most organized groups of dermatologists. In this society mutual good will is and always has been a distinctive feature, and it has enabled the society to function as an open forum harmoniously and with ever increasing effectiveness for these many years.

Dr. J. B. Herrick [2] commented as follows:

> Samuel Johnson once remarked that historical writing was a very inferior kind of literature . . . requiring no imagination and little invention, and fit only to be compared to poor poetry. . . . It is less true today [and only partially true for the time when it was made]. The historian . . . must write something other than a dreary record of dates or descriptions of battles, plottings of royalty or the amours of the courts.

In reviewing the historical records of this society, I find that there were no battles except diagnostic ones, no plotting except for progress and royalty in clinical perfection and no amours except brotherly love.

In a study of the records of this society and its progress, the sequence of dates and events takes on life because of the men who had to do with its origin and because of the loyalty of those who have carried on. At a meeting [3] on Dec. 5, 1911 the aims of the society were stated to be a "study of dermatology in all its branches, the edification of its individual members in their chosen specialty, and the cultivation of fraternal spirit among the members." That the society has superlatively achieved this is well known to all of us.

The organization of the Chicago Dermatological Society was the result of a movement which had been developing for several years among

---

President's address read before the Chicago Dermatological Society on Jan. 20, 1937.

1. Remarks by O. H. Foerster at annual dinner of Chicago Dermatological Society, Jan. 15, 1936 (Arch. Dermat. & Syph. **33**:746 [April] 1936).

2. Herrick, J. B.: William Allen Pusey—the Historian and Litterateur, Arch. Dermat. & Syph. **35**:14 (Jan.) 1937.

3. Recorded in the first public record of this Society (J. Cutan. & Genito-Urin. Dis. **21**:222 [May] 1913).

the active younger dermatologists of the city. The actual organization dates from a meeting held at the University Club on the evening of Feb. 14, 1901. On this occasion there were present Dr. James Nevins Hyde, who gave the dinner, and Drs. Joseph Zeisler, W. A. Pusey, H. G. Anthony, L. C. Pardee, W. L. Baum, L. B. Baldwin and F. H. Montgomery. Among others who were added to the list of charter members were Drs. Louis E. Schmidt, Howard T. Ricketts, David Lieberthal and Oliver S. Ormsby.[4] The first president of the society was Dr. Hyde.

The society was organized as a clinical one which should meet monthly from September to May inclusive and whose purpose should be the exhibition and discussion of cases. The first clinical meeting was held in Dr. Hyde's office on the afternoon of March 14, 1901, and meetings were held regularly for the remainder of the year. The society has faithfully followed its original purpose; monthly meetings have been held almost without failure for nine months of each of the years since its organization. At these meetings cases are presented, and after they have been examined general informal discussion of them takes place. The society has always been confined to active dermatologists, and so its meetings have been small. They began with meetings held in Dr. Hyde's or in Dr. Pusey's office; after Dr. Hyde's death Dr. Ormsby's office took the place of Dr. Hyde's. Except for one or two years when meetings were attempted in a rented room in an office building downtown, the meetings have always been held in private offices.[5] This has made them intimate and informal and has added distinctly to their usefulness to their participants.

The membership of the society has changed somewhat with the changes among the dermatologists of Chicago. Of the original members, Drs. Hyde, Zeisler, Anthony, Montgomery, Ricketts and Pusey[6] have died. They have all been eulogized in various issues of the ARCHIVES of DERMATOLOGY and SYPHILOLOGY; the most recent contribution of praise was Dr. Howard Fox's eloquent eulogy of Dr. Pusey in an editorial, "A Tribute to a Great Leader."[7] Two or three other members have left the city; but the personnel, particularly of active workers, except for death, has been little changed throughout the years of the existence of the society. The amount of clinical material shown at the meetings has been from the start surprisingly large. In fact, since the organization

4. Dr. Ormsby was in Europe at the time of the organization meeting.

5. Since April 1930 the clinical meetings have been held at the Cook County Hospital.

6. Dr. Pusey was present at the meeting at which this address was read. He died Aug. 29, 1940.

7. Fox, H.: William Allen Pusey, M.D., 1865-1940: A Tribute to a Great Leader, Arch. Dermat. & Syph. **42**:940 (Nov.) 1940.

of this society, and including the present meeting, there have been two hundred and sixty-one clinical meetings, at which over 3,500 clinical cases have been presented and discussed. There are few dermatoses instances of which have not been presented, and the value of the diffusion of clinical knowledge among its members which this rich exhibition of material has produced is hard to overestimate. A perusal of the transactions of the society, as published in the various editions of the ARCHIVES, will amplify this statement.

At the annual meetings there are usually twenty-five to thirty-five dermatologists from other cities in attendance, and this meeting has become an event in dermatology in the Middle West. The titles of the presidents' addresses given at the annual meetings of this society embrace practically every field of medicine and dermatology, and many of these addresses have been published in medical and dermatologic journals. The society presidents and their addresses [8] have been as follows: J. N. Hyde (1903)[9]; Joseph Zeisler (1904); H. G. Anthony (1905), The Attitude of the Medical Profession Regarding the Handling of Leprosy in Chicago; W. A. Pusey (1906)[10]; L. C. Pardee (1909), The Treatment of Ring Worm and Favus in School Children; D. Lieberthal (1910), The Use of Arsenic in Syphilis (the drug "606" was discussed by all present); O. S. Ormsby (1911), Pellagra: A Summary of the Work Done and the Findings of the Illinois Pellagra Commission [11] (Dr. Ormsby was secretary of this commission); O. H. Foerster (1912), Negative Wassermann Reactions in Tertiary Syphilis of Skin and Mucous Membranes (the treatment of acne was also the subject of general discussion at this meeting); E. L. McEwen (1913), Infantile Eczema (alopecia was also discussed); Joseph J. Zeisler (1914), The Lichen Group (there was also a discussion on autoserum therapy); F. G. Harris (1915), Etiology of Eczema; U. J. Wile (1916), Preliminary Report on Absorption of Mercury; W. A. Pusey (1917) was in Washington on business for the War Department, and the presidential address was given in 1918 by D. Lieberthal, vice president, whose subject was Bullous Infections of the Skin with Special Reference to Dermatitis Herpetiformis, Herpes Gestationes and Pemphigus Vulgaris; A. W. Stillians (1921); H. N. Cole (1922), Acute Syphilitic Transverse Myelitis; E. A. Oliver (1923); J. H. Mitchell (1924), Institutionalization of the Practice of Medicine [12]; E. P. Zeisler (1925); P. A. O'Leary (1926),

8. When the title of an address has been omitted, the available records have not given this information.

9. The first formal officers of the society were elected in 1903.

10. The first formal discussion of cases at an annual meeting was in 1906.

11. Ormsby, O. S.: Pellagra, J. Cutan. Dis. **30**:589, 1912.

12. Mitchell, J. H.: The Institutionalization of the Practice of Medicine, Illinois M. J. **48**:224 (Sept.) 1925.

Present Status of Nonspecific Treatment in Dermatology[13]; F. E. Senear (1927, 1928) (no address was given, since the annual meeting was a joint meeting with the Mississippi Valley Dermatological Conference); H. R. Foerster (1929); C. W. Finnerud (1930); H. E. Michelson (1931), Nodular Subepidermal Fibrosis[14]; M. H. Ebert (1932), Hypertrophic Striae Distensae[15]; O. S. Ormsby (1933); J. H. Mitchell (1934), Differential Diagnosis of the Acrodermatoses; W. A. Pusey (1935), The Field of Dermatology.[16] At the last-mentioned meeting resolutions[17] were presented to Dr. Pusey as retiring president by the members of the Chicago Dermatological Society as an "appreciative expression" for his faithfulness and influence on the society.

The presidents' addresses have covered varied clinical aspects of dermatology, the philosophic aspects of medicine and the elaboration of specific new advances in medicine and in dermatology that were of vital interest at the time the respective addresses were given.

This society has been host to the American Dermatological Association on two occasions, in December 1901 and in June 1933. It has been the privilege of the society to entertain the following visitors from foreign shores who have also participated in our clinical sessions: Dr. Norman Walker, Edinburgh, Scotland, May 17, 1920; Dr. J. M. H. McLeod, London, England, June 11, 1923; Dr. O. Gans, Heidelberg, Germany, Jan. 11, 1927; Dr. Bruno Bloch, Zurich, Switzerland, April 20, 1928, and Dr. J. Jadassohn, Breslau, Germany, June 21, 1929.

This essentially is the record of the Chicago Dermatological Society from its origin, as revealed by a study of the minutes and notes at hand. If, for any reason, a name has been omitted or proper credit has not been given where it was due, such omission is unintentional and is due to the fact that the exact information was not available at the time these notes were conceived.

Mrs. Irene H. Snyder, recording secretary of the society, gathered the various minutes and placed them at my disposal for study.

104 South Michigan Avenue.

---

13. O'Leary, P. A.: Present Status of Nonspecific Treatment in Dermatology, Arch. Dermat. & Syph. **15**:470 (April) 1926.

14. Michelson, H. E.: Nodular Subepidermal Fibrosis, Arch. Dermat. & Syph. **27**:812 (May) 1933.

15. Ebert, M. H.: Hypertrophic Striae Distensae, Arch. Dermat. & Syph. **28**: 825 (Dec.) 1933.

16. Pusey, W. A.: The Field of Dermatology: President's Address, Arch. Dermat. & Syph. **33**:987 (June) 1936.

17. Rattner, H.: William Allen Pusey at Close Range, Arch. Dermat. & Syph. **35**:25 (Jan.) 1937.

PSEUDOXANTHOMA ELASTICUM WITH ANGIOID STREAKS

THE SYNDROME OF GROENBLAD AND STRANDBERG

SEYMOUR H. SILVERS, M.D.
BROOKLYN
AND
HERMAN E. WOLFE, M.D.
NEW YORK

Although pseudoxanthoma elasticum with angioid streaks was first described as a syndrome by Groenblad and Strandberg,[1] the symptoms and signs were recognized and known by others before them. In 1889 Doyne [2] reported changes of the retina similar to those described later as angioid streaks. In 1892 Pflange [3] and Knapp [4] described such changes as angioid streaks of the retina. The first comprehensive description of the characteristic cutaneous lesions was that by Darier [5] in 1896. He described both the clinical and the histologic changes of the skin under the name of pseudoxanthoma elasticum. In 1904 Werther [6] called attention to the fact that pseudoxanthoma elasticum was a familial disease. Sugg and Stetson [7] reported the case of two sisters with pseudoxanthoma elasticum, angioid streaks and diabetes. Verhoeff [8] reported angioid streaks in a case of osteitis deformans. In his investigation of

From the Departments of Dermatology and Ophthalmology, Services of Drs. O. L. Levin and E. B. Gresser, Beth Israel Hospital, New York.

1. (*a*) Groenblad, E.: Angioid Streaks: Pseudoxanthoma Elasticum, Acta ophth. **7**:329, 1929. (*b*) Strandberg, J.: Verhandl. d. dermat. Gesellsch. **13**:3, 1929. (*c*) Groenblad, E.: Angioid Streaks: Pseudoxanthoma Elasticum, Acta ophth. (supp. 1) **10**:1, 1932.

2. Doyne, R. W.: Choroidal and Retinal Changes, the Result of Blows on the Eyes, Tr. Ophth. Soc. U. Kingdom **9**:128, 1889.

3. Pflange, O.: Pigment Stria with Secondary Changes in the Retina After Hemorrhage, Arch. Ophth. **21**:282, 1892.

4. Knapp, H.: On the Formation of Dark Angioid Streaks as an Unusual Metamorphosis of Retinal Hemorrhages, Arch. Ophth. **21**:289, 1892.

5. Darier, J.: Pseudoxanthoma elasticum, Monatsh. f. prakt. Dermat. **23**:610, 1896.

6. Werther: Ueber Pseudoxanthoma Elasticum, Arch. f. Dermat. u. Syph. **69**:23, 1904.

7. Sugg, E. S., and Stetson, D. D.: Pseudoxanthoma Elasticum Associated with Angioid Streaks of Retina and Diabetes Mellitus in Sisters, J. A. M. A. **102**:1369 (April 28) 1934.

8. Verhoeff, cited by Terry.[9]

22 cases of osteitis deformans, Terry [9] discovered 3 instances of angioid streaks. Vail [10] studied a case of pseudoxanthoma elasticum, angioid streaks and osteitis deformans.

The cutaneous and ocular lesions of this syndrome usually appear first during the third or fourth decade. Ohno [11] saw pseudoxanthoma elasticum in patients aged 10, 12 and 13 years respectively. In 1 of the cases he reported, the lesions were said to have existed since birth. There is a slight preponderance of males showing this syndrome. The onset of the cutaneous lesions is slow, and as they cause no subjective symptoms, the patient may be unaware of their existence for a long time. The ocular symptoms, however, are sudden in appearance. Retinal hemorrhages may cause sudden blurring in vision. The amount of vision lost depends on the extent of the hemorrhage. Although Doyne [2] reported that trauma preceded and probably was a factor in producing the retinal changes in his case, trauma was not stressed by subsequent observers as a factor in producing lesions of the eye.

REPORT OF A CASE

A. D., a man aged 46, born in Czechoslovakia, a cook, was admitted to the hospital complaining of loss of vision and generalized itching of three weeks' duration. His mother, aged 73, was living and well. The father died of heart trouble at the age of 60. Of his six brothers who were living, one was blind, supposedly as the result of his trade as a blacksmith. More details were not available, because the brother lived in occupied territory in Europe. The patient stated that he had never had syphilis. In 1919 he had an acute gonorrheal urethritis, which responded to treatment. His tonsils were removed in 1923, and a submucous nasal resection was performed. He was operated on in 1925 for a hernia and in 1932 for appendicitis. Twenty-five years ago, while he was living in a small village in Europe, his left eye became swollen and painful, and he was temporarily blind. With the use of home remedies and without medical consultation, the inflammation subsided, and vision returned but was somewhat diminished in the left eye.

Examination of the skin showed on both sides of the neck, extending upward to the face and downward to the clavicle, irregular, ill defined smooth patches having a velvety feel, devoid of hair and varying from light brown and yellow to shades of lilac, with some mottling. The follicular openings were more noticeable than on the normal skin and appeared as small macules. No definite papules were felt. Similar palm-sized patches were present in both axillas. They differed from the patches on the neck only in color. The patches in the axillas were chalk white. The difference in color in these areas was probably due to the fact that the lesions on the neck were constantly exposed to the elements, while the axillary lesions remained covered.

---

9. Terry, T. L.: Angioid Streaks and Osteitis Deformans, Tr. Am. Ophth. Soc. **32**:555, 1934.

10. Vail, cited by Terry.[9]

11. Ohno, T.: Pseudoxanthoma elasticum und dessen Histologie, Arch. f. Dermat. u. Syph. **149**:420, 1925.

A complete physical examination showed no defects other than those of the skin and the eyes. The blood pressure registered 130 systolic and 80 diastolic.

Examination of the eyes gave the following results: Uncorrected vision of the right eye was 20/100. The patient could only distinguish movements of the hand with the left eye. The muscular excursions were normal. The lacrimal apparatus was normal. The left cornea showed some superficial infiltration near the nasal limbus. The pupils were equal, round and regular and reacted to light and in accommodation. Examination of the fundi showed that the right disk

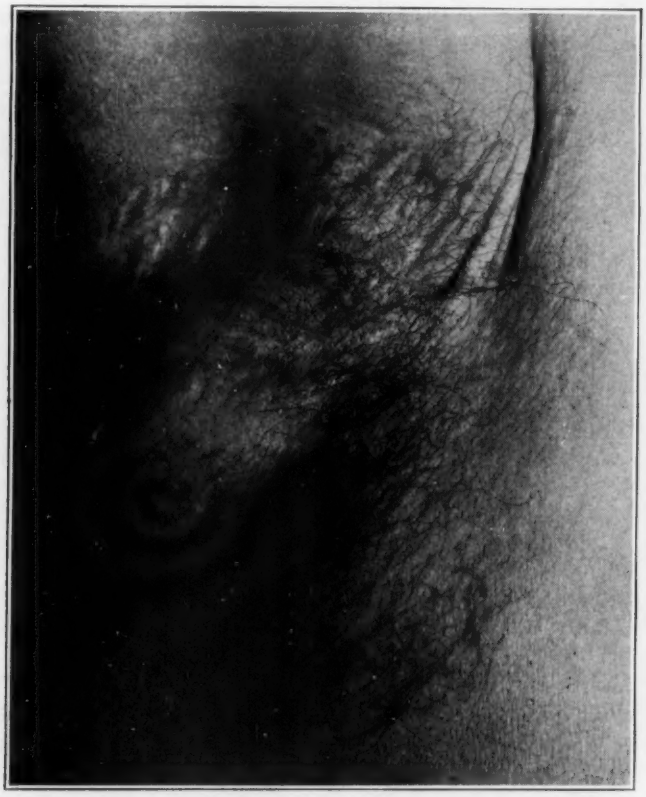


Fig. 1.—Pseudoxanthoma elasticum of the right axilla, showing raised papules on the upper margins.

was normal in color and outline. A network of arborization extended from the peripapillary area to the periphery. A flame-shaped hemorrhage was seen above the region of the macula. The left disk showed similar angioid streaks. The macula had an elevated area which was surrounded by a hemorrhagic wreath. Two rosette-shaped hemorrhages were present between the disk and the macula.

A histologic study of a section from lesions on the neck showed thinning of the epidermis and flattening of the rete pegs. In the subpapillary layer and in the midregion of the cutis the elastic tissue stain showed bundles of broken and fragmented elastic fibers. There was no inflammatory infiltration.

The reactions to Wassermann, Klein and Kahn tests of the blood were all negative. A Mantoux test with 0.001 mg. of tuberculin elicited a red swollen area 14 mm. in diameter in twenty-four hours. Intradermal tests showed that

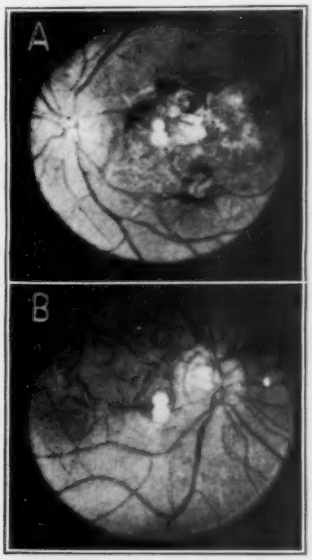


Fig. 2.—*A*, left fundus, showing hemorrhages resembling tufts of cotton; *B*, right fundus, showing less involvement than the left.

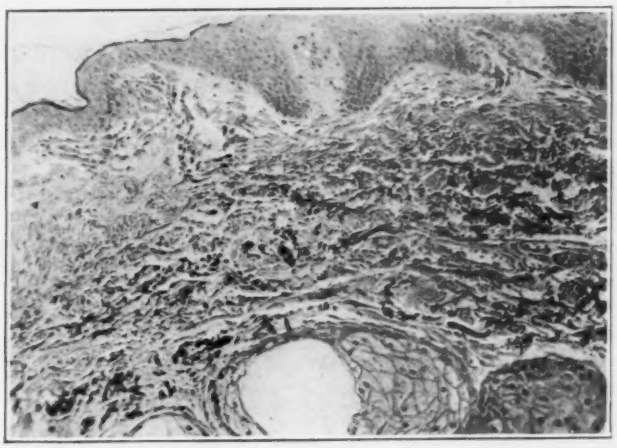


Fig. 3.—Section showing fragmentation of the elastic tissue (elastic tissue stain).

the patient reacted strongly to coffee and buckwheat and slightly to wheat, oranges, white potatoes, green peas, bananas, pecans, chocolate and peanuts. The sugar, urea, calcium, nonprotein nitrogen and cholesterol values of the blood were all within normal limits. Examination of the urine showed nothing abnormal. The

feces were free of ova and parasites. Two complete blood counts showed that all elements were within normal limits but that the eosinophils were 7 and 12 per cent, respectively, of the total white blood cell count. Roentgenograms of the long bones and of the skull showed no abnormalities. The arteries of the lower extremities showed extreme calcification.

COMMENT

The patient showed the classic signs and symptoms of the Groenblad and Strandberg syndrome. He was not aware of the onset of his eruption, since this was symptomless. He was rather definite about his eyes, as he remembered the sudden onset of diminished vision. This probably coincided with a severe retinal hemorrhage. Repeated examinations of the fundus always revealed new hemorrhages and absorption of old ones. His vision diminished progressively. The increased eosinophilic blood cell count was probably caused by the generalized itching.

It is apparent that in this syndrome the elastic tissue undergoes degenerative changes. Jadassohn [12] was unable to differentiate the degenerative changes of the elastic tissue from those of senile atrophy. Most observers at present are of the same opinion. Bloch [13] stated the belief that the degenerative changes of the elastic tissue were due to a toxin and thought that the eruption was more prevalent on the flexor surfaces because of continued stretching and bending. Freudenthal [14] stressed the familial character of the lesions and was inclined to classify them as nevi. In studying the autopsy observations in a case of this syndrome, Urbach and Wolfram [15] were impressed by the fact that the entire elastic tissue of the body showed degenerative changes similar to those seen in the skin. In their case the buccal mucosa was also involved. Although the systolic blood pressure in their patient had been only 110, the aorta showed diffuse dilatation and atheromatous plaques at autopsy. In the case herein reported, calcification of the arteries of the extremities was seen in the roentgenograms. Venkei [16] reported

12. Jadassohn, J.: Versammlung Schweiz. Dermat. Gesellsch. Dermat. Wchnschr. **63**:713, 1916.

13. Bloch, B.: Versammlung Schweiz. Dermat. Gesellsch. Dermat. Wchnschr. **63**:713, 1916.

14. Freudenthal, W., in Jadassohn, J.: Handbuch der Haut- und. Geschlechtskrankheiten, Berlin, Julius Springer, 1932, vol. 4, pt. 3 p. 435.

15. Urbach, E., and Wolfram, S.: Ueber Veränderungen des elastischen Gewebes bei einem autoptisch untersuchten Falle von Groenblad-Strandbergschem Syndrom, Arch. f. Dermat. u. Syph. **176**:167, 1937.

16. Venkei, T.: Pseudoxanthoma elasticum, Dermat. Wchnschr. **107**:1396, 1938.

2 cases of pseudoxanthoma elasticum in which the apparently normal skin also showed degenerative changes of the elastic tissue. According to this observer, the syndrome is due to a heredodegenerative process. One of his patients was syphilitic, while the other suffered from dementia paralytica. Finnerud and Nomland [17] demonstrated calcium deposits in the lesions of pseudoxanthoma elasticum.

SUMMARY

A case of pseudoxanthoma elasticum with angioid streaks is described.

The most likely explanation for the signs and symptoms of this syndrome is the one which stresses degenerative changes of the elastic tissue.

This syndrome is probably due to congenital defects.

The prognosis is poor, as ocular changes eventually cause blindness.

920 Bushwick Avenue, Brooklyn.

200 East Sixteenth Street, New York.

17. Finnerud, C. W., and Nomland, R.: Pseudoxanthoma Elasticum, Arch. Dermat. & Syph. **35**:652 (April) 1937.

COLLOID MILIUM

A VITAMIN DEFICIENCY DISEASE?

STUART C. WAY, M.D.
SAN FRANCISCO

Colloid degeneration of the skin, first described in 1866 by E. Wagner[1] under the name colloid milium, is an extremely rare disease. Besnier[2] reported a second case in 1879, to be followed by Feulard and Balzer[3] in 1885 and by Perrin,[4] who in 1892 presented the fourth instance of this interesting condition at the Second International Dermatological Congress, in Vienna.

More recently Jager,[5] in 1925, and Labadie,[6] in 1927, made valuable contributions to the subject, each presenting a case. Jager carefully reviewed the literature and was unable to find more than 20 undisputed examples of the condition as originally described by Wagner.

To the 22 cases already reported, another is being added, with the idea in view of ascribing to this condition a hitherto unmentioned etiologic factor.

REPORT OF A CASE

History.—S. T., a French-Basque man aged 45, a sheep herder, has lived for many years in the United States. His father died from a renal disease at the age of 60. The cause of the mother's death was unknown. Five sisters and two brothers were living and well. One brother was killed during the first world war. The patient has been tending sheep for many years near Ely, Nev. This occupation isolates him from civilization months at a time and requires exposure to intense heat during the summer and to extreme cold during the winter. The patient, who was first seen on Jan. 16, 1940, complained of small nodular lesions which involved the face and dorsa of the hands, areas exposed to the elements by the nature of his work.

---

1. Wagner, E.: Das Colloid—Milium der Haut, Arch. d. Heilk. **7**:463, 1866.

2. Besnier, E.: Etudes nouvelles die dermatologie, sur un cas de dégénérescence colloïde au derme, affection non décrite, non dénommée, ou improprement dénommée colloid-milium., Gaz. hebd. de méd. **16**:645, 1879.

3. Feulard, H.: Nouveau cas de dégénérescence colloïde du derme. Examen histologique par M. le Dr. Balzer, Ann. de dermat. et syph. **6**:342, 1885.

4. Perrin: Cas de dégénérescence colloïde du derme (colloïdome miliaire), Internat. Dermat. Cong. Ber. ü. I. Verhandl., 1892, p. 435.

5. Jager, T.: So-Called Colloid Degeneration of the Skin, Arch. Dermat. & Syph. **12**:629 (Nov.) 1925.

6. Labadie, J. H.: Colloid Degeneration of the Skin, Arch. Dermat. & Syph. **16**:156 (Aug.) 1927.

The present lesions first appeared on the forehead in 1928; they gradually spread laterally to the temporal areas, downward over the bridge of the nose and then to the cheeks, and later the dorsa of the hands became similarly affected. Subjective symptoms were completely absent.

An examination showed that these areas were covered with firm, shiny nodular elevations, varying in size from that of a pinhead to that of a buckshot. These

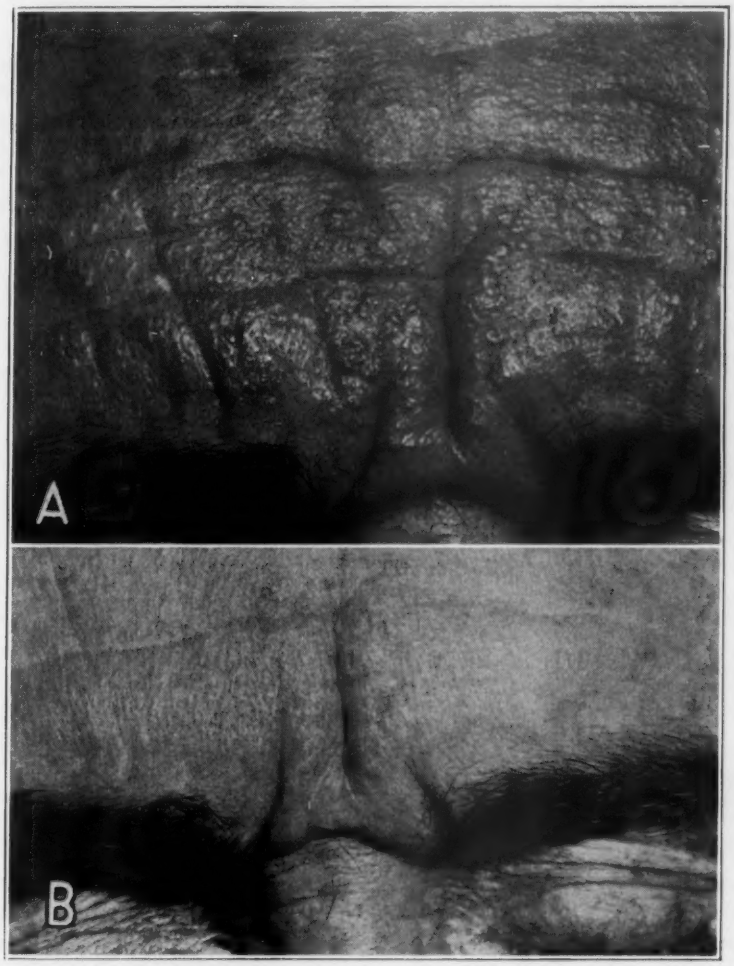


Fig. 1.—*A*, photograph taken on Jan. 16, 1940 shows numerous variously shaped and sized vesicle-like nodules on the forehead. *B*, photograph taken on April 14, 1941, after vitamin C therapy, shows great improvement.

vesiculoid, variously shaped nodules presented a yellowish skin color or translucent appearance and created the impression that they contained fluid. When punctured, a clear gelatinous material not unlike the contents of a mucoid or synovial cyst could be expressed.

Further examination revealed a small chalazion involving the left upper eyelid. The skin over other body parts appeared normal and consistent with the patient's age. No evidence of dermatophytosis existed.

Laboratory Examination.—The Wassermann and Kahn reactions were negative. Examination of the blood showed: hemoglobin, 89 per cent (Newcomer); color index, 0.91; erythrocytes, 4,910,000; leukocytes, 8,800; neutrophils, 67 per cent, of which 53 per cent were mature and 14 per cent stab cells; lymphocytes, 26 per cent; monocytes, 3 per cent, and eosinophils, 4 per cent.

The urine was normal. A spectroscope examination of the urine for hematoporphyrin showed the amount to be within normal limits, the faintest trace being found.

Histologic Studies.—A specimen for biopsy was taken from the dorsum of the left hand. The following staining methods were used: hematoxylin-eosin, Van

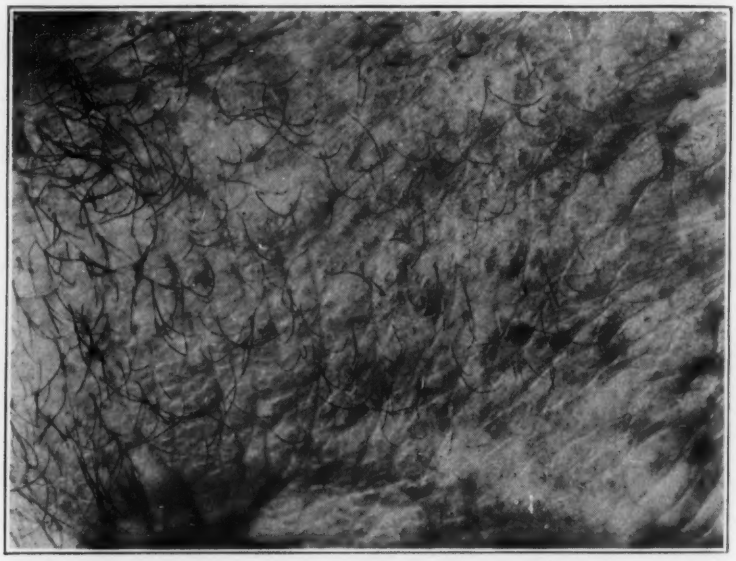


Fig. 2.—Dorsum of the hand, showing nodules similar to those on the forehead and nose.

Gieson, hematoxylin-orange "G," Weigert, orcein, polychrome methylene blue, elastin "H" hematoxylin iodine green and Unna's stain for collastin, collacin and elacin. As stated by other observers, a routine hematoxylin-eosin stain was found to be most satisfactory.

Histologic Examination.—The epidermis above the nodules appeared greatly thinned, owing probably to pressure from beneath and to the fact that the basal layer is included in the degeneration process. In many parts of the section, however, the epidermis appeared entirely normal, and the interpapillary processes were likewise unchanged. It was apparent that some of the degenerative changes occurred in the basal epidermal layer. In several areas, the cell boundaries were lost and disintegrated protoplasm together with the nuclei were being thrown out into the

adjoining corium. These degenerative changes within the basal layer are believed to occur above and simultaneous with the earliest microscopic evidence of a beginning colloid nodule.

The initial dermal changes appear to take place not within the papillary layer but around or within the walls of the subpapillary blood vessels. In several instances, the tunica externa of the blood vessels showed degenerative changes, and the endothelium appeared swollen. These changes were occasionally seen where little or no other evidence of disease existed and were accompanied by a slight perivascular round cell infiltration. Certain of the collagen fibers about the vessels were gradually becoming basophilic, as evinced by their staining ability and their wavy, fragmented and curled appearance, while others adjoining still took the eosin stain normally. The dermal layer showed various stages of the degeneration from the apparent normal collagen to the mature colloid nodule.

The elastic fibers, while definitely involved in the changes taking place, were less changed than the collagen and as a rule persisted in a fragmented form, singly or in clumps, even within the mature nodule. Throughout the reticular layer the elastic fibers appeared unchanged. The sweat and sebaceous glands also seemed normal.

Pathologic changes characteristic of the disease occupied the papillary and subpapillary layers and consisted of several cystlike areas, sharply demarcated from the rest of the tissues; these were composed of a homogeneous mass of material acidophilic in its staining ability, although less so than normal collagen. Throughout this finely granular mass numerous well preserved connective tissue nuclei could be seen. One nodule, mature in type, contained a few large cells with large oval nuclei. The cytoplasm of these cells so closely resembled the surrounding colloid that their borders could not be clearly seen. They occurred either singly or in groups and averaged about four or five times the size of a lymphocyte. Their appearance suggested that they were endothelial cells, probably survivors from partially destroyed blood and lymph vessels. Above, the colloid mass was separated from the epidermis by a narrow band of connective tissue fibers; while laterally and below, it was surrounded by a dense network of elastic fibers which appeared swollen and broken down into short segments, of which many were fused but retained their specific staining ability.

The microscopic observations closely conform with those in previously reported cases. All of the characteristic histologic features were observed, even those of Bosellini,[7] who was the only writer to find evidence that the cells of the epidermis shared in the degeneration. The sections studied, however, failed to confirm his observation that some of the cells of the rete malpighii present a homogeneous appearance, as if they had been penetrated by colloid substance. In this case, however, disintegration of the basal cells characterized the earliest epidermal change. Simultaneous changes in the cutis, first noted by Balzer [8] and confirmed by later writers, appear to occur within the vessel wall and were present in this case.

---

7. Bosellini, P. L.: Sur deux cas de pseudo-milium colloïde familial, Ann. de dermat. et syph. **7**:751, 1906.

8. Balzer, cited by Feulard.[3]

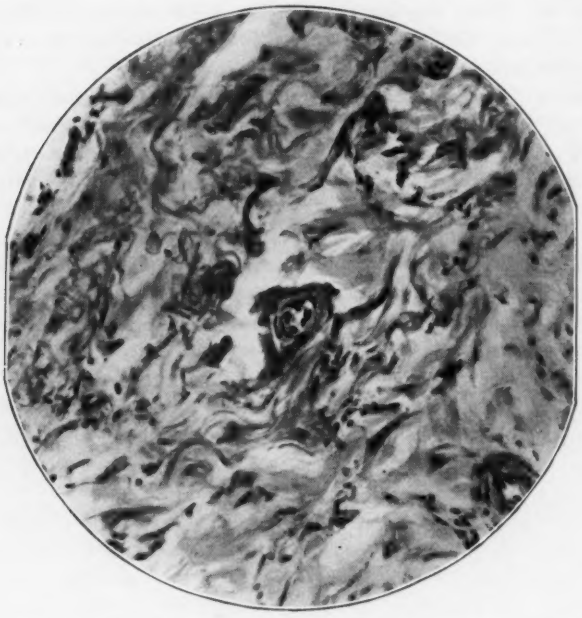


Fig. 3.—Section from the dorsum of the hand, showing early vascular and perivascular degenerative changes (hematoxylin-eosin stain).

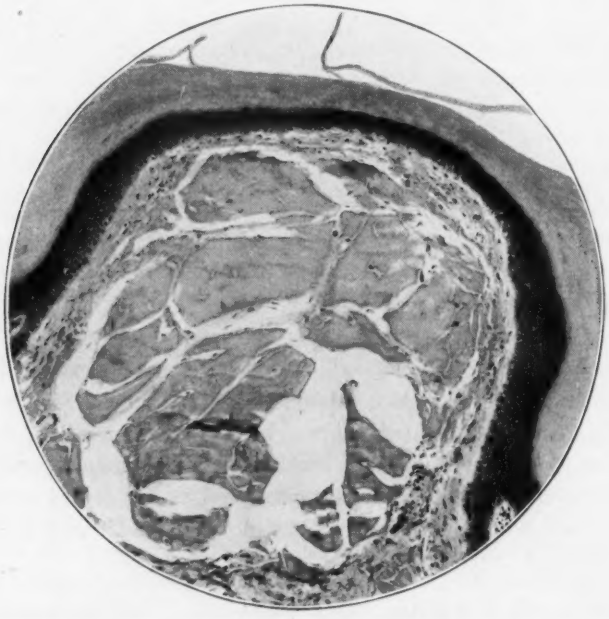


Fig. 4.—Section of a mature nodule on the dorsum of the hand, showing fragmentation of its contents (hematoxylin-eosin stain; × 70).

The cells with large nuclei found within the colloid mass were similar to those already described by Bizzozero [9] and Ketron.[10] It is extremely doubtful whether one is justified in regarding them as "macrophags"; perhaps "inclusion cells" would be a better term, as they were probably the cellular remains of destroyed vessels.

While the elastic fibers were definitely involved in the degenerative changes, the scarcity of fibers mentioned by other writers could not be confirmed, as they persisted, although greatly decreased in number, even within the colloid masses.

The term colloid is used to represent a product of cell activity or degeneration. Although the name was originally applied to the peculiar transparent jelly-like substance of the thyroid gland, its loose usage makes uniformity of opinion difficult. Klebs [11] regarded colloid degeneration as a degeneration in which homogeneous masses were deposited similar to thyroid colloid, and he included under the heading of albuminous degenerations, colloid, amyloid, hyaline and mucoid degenerations. Ernst [12] likewise showed that the colloid which resulted from degeneration of connective tissue differed in its staining reaction from thyroid colloid, since with Van Gieson's method the degenerative product stained red while thyroid colloid stained yellow. Macleod and Muende [13] regarded colloid as a homogeneous structureless substance which occurs in refractile masses or blocks resulting from the degeneration of the fibrous elements of the corium and which is neither amyloid nor glycogen but a complex albuminous substance whose chemical nature is as yet undetermined.

Unna's opinion that collastin, a degenerative product of collagen, becomes transformed into colloid [14] must still be accepted. Although degeneration of elastin occurs, the presence of apparently normal fibers (except for shape) within the mature colloid material makes it extremely doubtful that they form an important constituent part.

Vitamin Studies.—The patient was presented on Feb. 17, 1940 at a joint meeting of the Los Angeles and San Francisco dermatologic societies [15] in order to invite

9. Bizzozero, E.: Ueber eine klinisch ganz eigenartige Form von pseudocolloid Milium, Arch. f. Dermat. u. Syph. **95**:35, 1909.

10. Ketron, L. W.: A Case of Colloid Degeneration of the Skin with Unusual Histological Features, Bull. Johns Hopkins Hosp. **29**:163, 1918.

11. Klebs, E.: Allgemeine Pathologie, oder die Lehre von den Ursachen und dem Wesen der Krankheitsprocesse, Jena, Gustav Fischer, 1889, vol. 2, p. 377.

12. Ernst, P.: Ueber Hyalin, insbesondere seine Beziehung zum Colloid, Virchows Arch. f. path. Anat. **130**:377, 1892.

13. Macleod, J. M. H., and Muende, I.: Pathology of the Skin, ed. 2, New York, Paul B. Hoeber, Inc., 1940, p. 155.

14. Unna, P. G.: The Histopathology of the Diseases of the Skin, translated by N. Walker, New York, Macmillan & Co., 1896, p. 988.

15. Way, S. C., and Haag, E. C.: Hyaloma (Colloid Degeneration), Arch. Dermat. & Syph. **43**:408 (Feb.) 1941.

discussion and obtain therapeutic suggestions. The use of ascorbic acid (vitamin C) was suggested by Dr. Nelson P. Anderson.[16] Its use seemed worthy of trial, as the patient on account of his isolation went without fruit and fresh vegetables for months at a time.

The total volume of urine for twenty-four hours was 880 cc. The total amount of ascorbic acid excreted was 2.2 mg. (the normal daily output varies between 10 and 30 mg.). Then 500 mg. of ascorbic acid was administered orally. A total of 910 cc. of urine for twenty-four hours contained 9.1 mg. of the substance. The subnormal quantity excreted and the large amount retained by the body suggested a vitamin C deficiency.

The patient before returning to Nevada was given a diet high in vitamin C and was also given 50 mg. capsules of ascorbic acid, which were to be taken by mouth three times daily.

After an absence of ten months, the patient returned to San Francisco for further observation. His dislike for medicine caused him to discontinue the internal medication within two months after it had been prescribed, but foods high in vitamin C were still being eaten in large quantities. The improvement that had occurred is clearly shown by the photographs. The hands alone presented sufficient pathologic changes for a clinical diagnosis to be made.

Regardless of his high vitamin diet, the patient's blood serum at this time showed only 0.61 mg. of ascorbic acid per hundred cubic centimeters, as compared with a normal value of 0.7 to 1.4 mg.

COMMENT

Recent clinical and laboratory experimentation tends to support the contention that vitamin C is an important factor in regulating the development of collagen. Lanman and Ingalls[17] performed laparotomy on guinea pigs which had been on previous scorbutic diets. At intervals after operation the animals were killed, and the authors reported that the wounds broke down more readily in the scorbutic pigs than in the nonscorbutic controls. On histologic examination they noted defective repair of the corium and a poor production of collagen in the scars of the scorbutic animals. This is in accord with the role that vitamin C is supposed to play in the formation of collagen.

Dalldorf[18] stated that because vitamin C is assumed to have a pronounced effect on oxidation-reduction phenomena in the tissues and because hydrogen ion concentration is of critical importance to colloidal systems, it is tempting to accept the simpler explanation that the vitamin directly alters the physical character of the intercellular fluid.

Defective intercellular substances are seen in connective tissue, bones and teeth and are believed to occur in the blood vessels. The early degenerative vascular and perivascular changes in colloid milium are

16. Anderson, N. P.: Personal communication to the author.

17. Lanman, T. H., and Ingalls, T.: Vitamin C Deficiency and Wound Healing: Experimental and Clinical Study, Ann. Surg. **105**:616, 1937.

18. Dalldorf, G.: The Pathology of Vitamin "C" Deficiency, in The Vitamins, Chicago, American Medical Association, 1939, p. 389.

more easily understood when one remembers that capillaries develop from embryonic connective tissue. The endothelium of capillaries is believed to be fused together by a cement substance; but the capillary is also accompanied and surrounded by connective tissues, and thin collagenous fibers ensheath the endothelium. Whether the effect of the vitamin deficiency is on the sheath or on the endothelial cement substance is still unknown.

If, therefore, colloid milium can rightfully be placed among the avitaminoses, it is a much rarer disease than one would expect, owing perhaps to its lack of recognition by the general practitioner, as in the present case. Previous writers have agreed that its cause is unknown. Wagner [1] considered the disease primary in the sebaceous glands and therefore assigned the name colloid milium, but this impression was later corrected by Besnier [2] and Balzer.[3]

The predisposing factors in all of the reported cases show an unmistakable similarity. Overexposure to the elements has been mentioned. This patient just described, who was a sheep herder, lived in Nevada, which is subject to extremes of temperature; therefore more than ordinary exposures to heat and cold occurred. As other members of his family resided in Europe, it cannot be stated whether their skins would have reacted in a similar manner under the same living conditions.

In the majority of reported cases the condition occurred in males, which is to be expected, as they are subject to greater hardships. The age limits have varied from 7 to 57 years. No seasonal variation occurred, such as was noticed in Jager's case.[5] Many of the observers stated the belief that this disease is essentially a chronic one which persists for years, although involution has been noted in 3 cases.

Colloid milium like pellagra, a vitamin deficiency disease, occurs mostly in rural districts among the poor, who eat an overabundance of grain products and an insufficient amount of animal protein, fruits and vegetables. The lesions also show a predilection for the uncovered parts of the body and usually involve the forehead, face and dorsa of the hands. The conjunctiva and mucous membrane have also been affected.

However, hematoporphyrin, which is frequently present in pellagra, was found to occur within normal limits in this patient. Exposures to actinic rays are believed to be a predisposing factor in both diseases.

SUMMARY AND CONCLUSIONS

Another typical example of colloid milium of Wagner has been reported.

The vitamin C deficiency existing in this case and the improvement seen following its attempted correction suggest that further studies may place this disease among the avitaminoses.

490 Post Street.

PIGMENTATION OF THE SKIN DUE TO VITAMIN A DEFICIENCY

REPORT OF A CASE AND REVIEW OF THE LITERATURE

JESSE A. TOLMACH, M.D.
AND
THOMAS N. GRAHAM, M.D.
NEW YORK

Although the effects of vitamin A deficiency on the growth of the body and specifically on the eye have been recognized for more than twenty years, changes in the skin have been noted only within the past few years. We wish to report the following case in which there are lesions of the skin which we believe are due to a deficiency in vitamin A.

REPORT OF CASE

L. N., a woman aged 40, of Irish and Egyptian parentage, was first examined on June 9, 1939. She complained of an abnormal brown pigmentation which had been present on the wrists for one year and on the knees for three months.

The past history was irrelevant. The patient's father had died of "kidney trouble"; her mother had cardiac disease and hypertension and was still living. There was no complaint of gastrointestinal crises or asthenia. The patient had been depressed over her mother's illness and had suffered from anorexia for several months prior to the onset of the eruption. Her diet had been particularly lacking in vegetables and fruits. The thyroid gland was slightly enlarged. The blood pressure was 132 systolic and 74 diastolic. Otherwise the observations on physical examination were of no importance. There was no apparent evidence of Addison's disease.

Dermatologic examination showed a generally dry skin. On the flexor and extensor surfaces of the right forearm and on the flexor surface of the left forearm there were many perifollicular pinpoint-sized to pinhead-sized brown pigmented spots. There were a number of black follicular keratotic plugs over the dorsum of the first metacarpal bone of the right hand (fig. 1). Similar plugs were present on the dorsal surfaces of the middle phalanges of the third, fourth and fifth fingers of both hands. The anterolateral surfaces of the thighs and the anterior surfaces of the legs showed innumerable pinpoint-sized to pinhead-sized discrete perifollicular depositions of pigment, and in these areas there were many follicular keratoses. The brown pigmentation was most pronounced on the anterior and lateral surfaces of the right knee (fig. 2*A*). In the latter area the pigmented spots were confluent and a deeper brown with a suggestion of a violaceous tinge. There were a number of discrete brown papules in this patch. On the left knee there was a similar but less pronounced eruption. The sides of the neck and the

lateral surface of the abdomen showed pale pigmented lesions of the same type. There was no pigmentation of the buccal mucosa.

A thorough ophthalmologic examination revealed only some choroidal pigmentation about the disk, which was not considered abnormal.

The urine was normal. A vitamin C saturation test showed 0.8 mg. of ascorbic acid per hundred cubic centimeters of plasma and a total of 565 mg. for five hours' excretion following the administration of 1,000 mg. intravenously. The determinations of sodium and potassium in the serum were normal. The Wassermann reaction of the blood was negative.

A histologic study of sections from a specimen of skin taken from the right knee showed an atrophic epidermis with edema and swelling of many of the basal cells and edema between the corium and the epidermis. There were small islands of round cells about the distended blood vessels. Throughout the entire upper part of the corium there were numerous cells containing a brown pigment. The lower

Fig. 1.—The right hand of a patient with vitamin A deficiency before the institution of vitamin A therapy, showing the presence of the dark follicular plugs and the areas of increased pigmentation.

margin of the cellular exudate ended abruptly at the level of the upper part of the corium. A section stained with methylene blue showed a black pigment through the upper part of the corium.

A Van Gieson stain showed fine strands of yellow between the collagen fibers but not more than normal. A Weigert stain showed the presence of the pigment and swelling of the elastic tissue fibers. There was little change at the openings of the hair follicles. The scale did not thicken to any extent at the follicular openings. A melanin bleach showed absolutely no pigment, and the Perl stain showed no evidence of iron pigment.

A specimen of skin from the right hand showed widening of the follicular openings. There was no atrophy in this area except at the sides of the hair follicles, even though the openings were filled with horny plugs. There was a perivascular cellular reaction, most evident in this area. Brownish pigment was scattered throughout the upper part of the corium, most noticeable where the exudate was least intense.

On July 7 examination with a visual adaptometer or biophotometer showed a delay in dark adaptation suggestive of vitamin A deficiency. Vitamin therapy was instituted, and after two months of treatment a second examination with the same apparatus was made, in November 1939. At this time great clinical improvement was evident and the adaptometer showed a normal adaptation curve. These observations must be considered inconclusive, however, in view of the fact that the fixation was not controlled. The area tested could not be determined with the type of apparatus employed.

Fig. 2.—The anterolateral aspect of the right knee, showing (*A*) the intense pigmentation and the dark follicular keratoses due to vitamin A deficiency and (*B*) the diminution of the pigmentation after ten weeks of vitamin A therapy.

On October 9 the patient was placed on a diet rich in vitamin A and was given capsules of halibut liver oil, amounting to 25,500 U. S. P. units of vitamin A daily. Three weeks later the follicular keratoses were definitely less evident and the pigmentation was slightly lighter. The skin was softer and less dry. Seven weeks after the institution of vitamin A therapy, the keratotic lesions had entirely disappeared, and the pigmentation in all areas had decreased obviously. By the tenth week of treatment the pigmentation had been reduced at least about 50 per cent in intensity (fig. 2 *B*). The vitamin A therapy was continued for a total

of fifteen weeks, but after the tenth week there was no further diminution in the pigmentation. The vitamin A treatment was then stopped, and the patient's diet was no longer rigidly supervised. Within two weeks there was evidence of a recurrence of the follicular keratoses. When she was seen eleven weeks after treatment was stopped, she showed a further increase of follicular keratoses, particularly on the anterior surfaces of the thighs and on the extensor and lateral surfaces of the forearms. The keratoses present then were much less pronounced than those seen originally. There appeared to be only a slight increase in pigmentation.

REVIEW OF THE LITERATURE

It is well known that xerophthalmia, keratomalacia, night blindness and cutaneous changes may occur as a result of vitamin A deficiency. Further investigation is necessary to confirm the many clinical reports of involvement of other organs in this disease.

In regard to the skin, Frazier and Hu [1] pointed out that a deficiency of vitamin A results in definite alterations in the cutaneous surface. They described a series of cases in which there was a symmetric eruption involving, in order, the anterolateral parts of the thighs, the posterolateral surfaces of the upper parts of the forearms, the extensor surfaces of the upper and lower extremities, the shoulders, the abdomen, the back, the buttocks and finally the posterior aspect of the neck. In these areas there was hyperkeratosis especially of the hair follicles, in the form of dry, firm, pigmented papules containing central intrafollicular keratotic plugs. Many of these plugs projected from the hair follicles as horny spines or were covered with a loosely adherent scale. When expressed, the plugs left gaping cavities. The papules were conical to hemispherical. In some patients the skin was generally darker than normal, and in a few it was a dull slate color. There was frequently absence of sensible sweating, and the skin was dry and harsh. These authors described the pathologic process as consisting primarily of hyperkeratinization of the lining epithelium of the hair follicles. They found also hyperplasia of the epidermal cells adjacent to the hair follicles in the papular areas where there was also an increase of intracellular pigmentation. There was an absence of sebaceous glands adjacent to the damaged follicles, and the sweat glands and ducts were involved. They stated the belief that hyperkeratosis of the hair follicles in association with xerophthalmia was a specific character of vitamin A deficiency and that the skin may present the only clinically detectable manifestation of the disease.

1. Frazier, C. N., and Hu, C. K.: Nature and Distribution According to Age of Cutaneous Manifestations of Vitamin A Deficiency: Study of Two Hundred and Seven Cases, Arch. Dermat. & Syph. **33**:825 (May) 1936.

Reports pertaining to this dermatosis were made also by Loewenthal,[2] Nicholls,[3] Reiss,[4] Mitchell,[5] Youmans and Corlette,[6] Sweet and K'ang,[7] Lehman and Rapaport [8] and others.

According to Mu, Frazier and Pillat,[9] the skin and conjunctivas of patients with vitamin A deficiency contain both melanin-building ferments and melanin pigment, as shown by the positive dopa and silver reactions. In the skin the dopa and silver reactions are both decreased in the epidermis around the hyperkeratotic hair follicles and increased in the adjacent hyperplastic epithelial processes. On the other hand, Youmans and Corlette failed to find pigmentation in white persons. Our patient was a white woman, and pigmentation was the outstanding feature. Goldsmith [10] pointed out that deficiency in vitamin A in man may cause, in addition to dryness and papular lesions, intense pigmentation. He also speculated as to the possibility of a relation between vitamin A deficiency and the pigmentation seen in vagabonds when there is also involvement of the conjunctivas.

In the cases reported by Frazier and Hu, Loewenthal and most other observers, the symptoms of vitamin A deficiency were caused by an insufficient amount of this vitamin in the diet. There is, however, the possibility that a deficiency of vitamin A may arise in association with chronic diarrhea, obstruction of the biliary ducts, pancreatic dysfunction, celiac disease and other diseases involving prolonged faulty fat metabolism.[11] Wilbur and Eusterman [12] reported a case of vitamin A deficiency in which the condition resulted from faulty absorption and was corrected after repair of a gastrocolic fistula.

---

2. Loewenthal, L. J. A.: New Cutaneous Manifestation in Syndrome of Vitamin A Deficiency, Arch. Dermat. & Syph. **28**:700 (Nov.) 1933; East African M. J. **15**:58 (May) 1933.

3. Nicholls, L.: Indian M. Gaz. **68**:681 (Dec.) 1933.

4. Reiss, F.: Chinese M. J. **50**:945 (July) 1936.

5. Mitchell, J. P.: East African M. J. **10**:38 (May) 1933.

6. Youmans, J. B., and Corlette, M. B.: Am. J. M. Sc. **195**:644 (May) 1938.

7. Sweet, L. K., and K'ang, H. J.: Clinical and Anatomic Study of Avitaminosis A Among Chinese, Am. J. Dis. Child. **50**:699 (Sept.) 1935.

8. Lehman, E., and Rapaport, H. G.: Cutaneous Manifestations of Vitamin A Deficiency in Children, J. A. M. A. **114**:386 (Feb. 3) 1940.

9. Mu, J. W.; Frazier, C. N., and Pillat, A.: Chinese J. Physiol. **11**:247 (March) 1937.

10. Goldsmith, W. N.: Recent Advances in Dermatology, Philadelphia, P. Blakiston's Son & Co., 1936.

11. Altschule, M. D.: Vitamin A Deficiency in Spite of Adequate Diet in Congenital Atresia of Bile Ducts and Jaundice, Arch. Path. **20**:845 (Dec.) 1935.

12. Wilbur, D. L., and Eusterman, G. B.: Nutritional Night Blindness: Report of Case, J. A. M. A. **102**:364 (Feb. 3) 1934.

In order to confirm the diagnosis of vitamin A deficiency it would, of course, be most helpful were there some reliable and accurate laboratory method. Determinations of vitamin A in the blood have been made, but hardly enough work has yet been done properly to evaluate their significance. Although a number of studies have been made, there has not been any uniformity in the methods used by the various investigators. Steininger, Roberts and Brenner,[13] in a carefully controlled study, showed that consistently low fasting blood values indicate the probability that the body stores of vitamin A are being depleted and that high values indicate that the intake of vitamin A is adequate. They stated, however, that the evidence as to whether or not determinations of the vitamin A in the blood are of value in judging nutritional status is contradictory. Their results further showed that there is no correlation between the amount of vitamin A in the blood and the biophotometer readings.

Dark adaptation tests with the biophotometer for the determination of vitamin A deficiency have received a great deal of attention. Fredericia and Holm [14] and Holm [15] observed that the rate of regeneration of the visual purple after its bleaching by a bright light in the living animal was less in rats receiving a diet deficient in vitamin A than in the controls. Yudkin, Kriss and Smith [16] showed that the fat extracted from the retina is one of the richest sources of vitamin A and suggested that visual purple may bear a direct structural relation to vitamin A. Jeans [17] observed that 25 per cent of rural children and 53 per cent of city children showed poor visual adaptation and that they were cured in four to six weeks with the administration of cod liver oil or of carotene equivalent to 5,000 to 6,000 U. S. P. units of vitamin A daily. Snelling [18] disagreed with the reliability of the biophotometer readings. Palmer and Blumberg [19] have shown that improvement in visual acuity with repetition of the test may be due to practice and that variations among normal children are so great that the results of single tests are unreliable. Isaacs, Jung and Ivy [20] failed

13. Steininger, G.; Roberts, L. J., and Brenner, S.: Vitamin A in Blood of Normal Adults: Effect of Depletion Diet on Blood Values and Biophotometer Readings, J. A. M. A. **113**:2381 (Dec. 30) 1939.

14. Fredericia, L. S., and Holm, E.: Am. J. Physiol. **73**:63 (June) 1925.

15. Holm, E.: Am. J. Physiol. **73**:79 (June) 1925.

16. Yudkin, A. M.; Kriss, M., and Smith, A. H.: Am. J. Physiol. **97**:611 (July) 1931.

17. Jeans, P. C.: Minnesota Med. **16**:688 (Nov.) 1933.

18. Snelling, C. E.: J. Pediat. **9**:655 (Nov.) 1936.

19. Palmer, C. E., and Blumberg, H.: Pub. Health Rep. **52**:1403 (Oct. 8) 1937.

20. Isaacs, B. L.; Jung, F. T., and Ivy, A. C.: Vitamin A Deficiency and Dark Adaptation, J. A. M. A. **111**:777 (Aug. 27) 1938.

to find any correlation between the intake of vitamin A and the readings made with the instrument used by Jeans. Clausen [21] stated that further observations are necessary in order to establish the value of tests of adaptation in dim light as a criterion of the reserves of vitamin A in the body. Hecht,[22] however, stated the belief that measurements of dark adaptation when made under properly specified conditions can be used as an aid in the diagnosis of the vitamin A condition of the body and that normal dark adaptation means that the vitamin A content of the body is normal. He admitted that the term normal still remains to be precisely defined but for the present accepted it in its ordinary usage. He further stated that abnormal dark adaptation indicates that the subject's retina is not adequately supplied with vitamin A, unless he possesses an inherited hemeralopia or his cone function is congenitally deficient. When such genetic factors have been excluded, it becomes necessary to investigate the basis for the retinal lack of vitamin A. If a faulty diet is alone responsible, treatment is obvious and response rapid. There may, however, be some metabolic deficiency which, in spite of an adequate diet, is responsible for an insufficient deposit of vitamin A in the retina. Cases of this type require thorough study.

COMMENT

Our patient's eruption became much less pronounced during a period in which treatment was limited to 25,500 U. S. P. units of vitamin A daily and an increase in quantity of her usual diet, which included a sufficient proportion of foods rich in vitamin A. This obvious improvement and the exacerbation which occurred when treatment was discontinued, we believe, make a diagnosis of vitamin A deficiency most probable.

Although the biophotometric readings obtained with the instrument we used may be of questionable accuracy, it is interesting to note that our readings were consistent with the clinical course.

The cause of the vitamin A deficiency is not clear in this case. Although the patient's diet had been well balanced, her meals had been irregular and the amount of food inadequate. We believe, however, that other factors are involved, in view of the exacerbation of the eruption when vitamin A therapy was discontinued and the patient remained on an adequate diet. It would seem possible, then, that there may have been some factors interfering with the absorption and assimi-

21. Clausen, S. W.: The Pharmacology and Therapeutics of Vitamin A, in The Vitamins: A Symposium, Chicago, American Medical Association, 1939, p. 60.

22. Hecht, S.: Nutrition: The Newer Diagnostic Methods, read at the Sixteenth Annual Conference of the Milbank Memorial Fund, New York, March 29-31, 1938.

lation of the carotene and vitamin A in the food ingested. Although no cause for interference with absorption and assimilation has as yet been demonstrated in our patient, the possibility must be considered. We shall continue our investigation in this regard.

The next point of interest in this case is the extensive irregularly distributed brown pigmentation, which feature is not emphasized by most observers. Frazier and Hu, Loewenthal and others have reported a not unusual darkening of the skin and the presence of pigmented follicular papules. As mentioned before, Goldsmith referred to the possibility of intense pigmentation in cases of vitamin A deficiency. A glance at the photographs will indicate the intensive pigmentation which was present, especially over the right knee. Mu, Frazier and Pillat have shown that there is an increase of melanin-building ferments and melanin pigment in the hyperplastic epithelial processes adjacent to the hyperkeratotic hair follicles in cases of vitamin A deficiency.

The pigmentation did not disappear entirely as did the follicular keratotic lesions under vitamin A treatment. The reduction in intensity of the pigmentation was, however, evident. We are not certain whether, after fifteen weeks of this treatment, we reached a permanent residuum of pigmentation or whether coexisting causative factors continued to operate.

SUMMARY

A case of intensive brown pigmentation of the skin associated with follicular keratoses, which we believe is probably due to vitamin A deficiency, is described.

Treatment with 25,500 U. S. P. units of vitamin A daily resulted in marked diminution of the pigmentation and disappearance of the follicular keratoses.

The clinical picture may have been partially or entirely produced by a deficiency in absorption and assimilation of vitamin A from the patient's diet.

Biophotometric readings were obtained in this case. Although the changes in dark adaptation seem to correspond with the clinical course, these readings are not offered as conclusive evidence because of the questionable accuracy of the apparatus we employed.

Miss Lucille Sobacke, Associate Director of Orthoptics at the New York Eye and Ear Infirmary, performed the biophotometer tests.

30 West Fifty-Ninth Street.

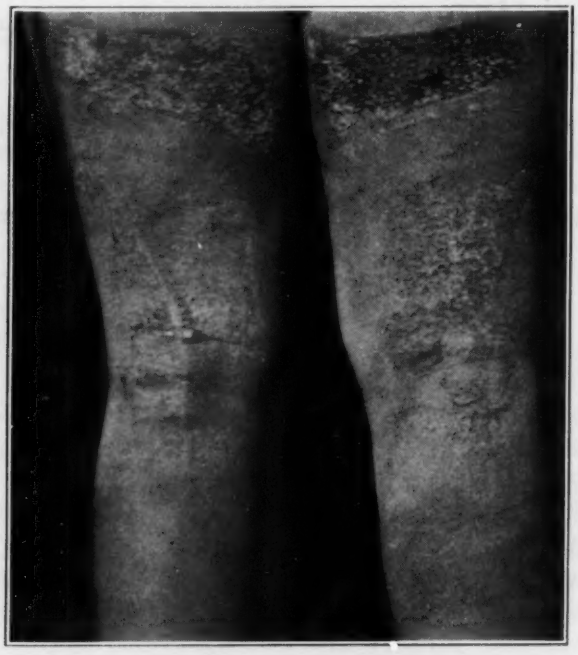
118 East Sixty-First Street.

Clinical Notes

DERMATITIS FROM IMITATION LEATHER

SEYMOUR J. WHITE, M.D., LAWRENCE, MASS.

Mrs. M. L. was referred to me for an acute dermatitis of two days' duration. Examination disclosed symmetric sharply demarcated areas on the middle third of the posterior aspect of each thigh. The affected areas presented an acute vesiculobullous dermatitis. There was no clear skin within the involved areas.



Dermatitis from imitation leather.

Similar lesions were present in the popliteal spaces, extending up the thigh and down the leg; the proximal delimitation was abrupt and straight lined, and the distal margin was slightly irregular and faded gradually into the normal skin. All the lesions ceased abruptly at the lateral margins of the affected areas. Extending in a curved line from the proximal external angle of the lower lesion on each thigh and ending mesially at the lower border of the dermatitis was a narrow band of normal skin, about 2 mm. in width. The rest of the skin was normal except for a patch of erythema with papules and vesicles on the dorsum of the right hand and wrist.

Two years before the patient had had an axillary dermatitis from a deodorant. The rest of the past history was irrelevant.

The immediate history excluded any contact with new or recently cleansed, dyed or painted material. However, further questioning along this line elicited the information that itching of the affected areas had preceded the appearance of the rash on the morning following a visit to the moving pictures. The patient had worn a short dress cut in such fashion that it pulled up considerably when she was seated. She was then examined wearing the same dress and in the seated position. It was immediately obvious that the sharply outlined symmetric plaques coincided with a bared area of skin. The clear area immediately distal to these plaques corresponded to the point of contact of the reenforced tops of the stockings. Other lesions were present where the sheer stocking began. The clear line extending down throughout the dermatitis was explained by the thick seam of the stocking. The lesion on the right hand and wrist corresponded to the area of skin which came in contact with the chair when the dress was smoothed as the patient seated herself. It was learned that in the moving picture theater all the chairs had recently been recovered with imitation leather. A sample of this covering was supplied for testing.

Patch tests were made on the patient's arm with both sides of the material. Eighteen hours after application there was intense itching at the site of the tests. On removal of the patches at the end of twenty-four hours, there was an erythematous area exactly the size and shape of the material applied, covered with a large bulla. The test with the reverse side of the material elicited slight erythema at the end of twenty-four hours, when it was removed. Two days later there was a reaction similar to the aforementioned one, except that it was more severe at the periphery of the test site.

Four healthy persons were given patch tests with both sides of the imitation leather. All reactions were negative.

This case is reported because of the rarity of the condition and its proved origin as a dermatitis from limited (three hours) contact with an imitation leather chair. Lesions developed not only on the bare skin which was directly exposed but also on areas covered with silk stockings. The skin which was covered by the reenforced tops and the seams of the stockings was not affected.

Extreme sensitivity is indicated by the fact that the dermatitis involved the right hand and wrist after limited contact. Patch tests with both sides of the imitation leather elicited strongly positive reactions. As far as I have been able to ascertain, no other similar cases have been reported in the community.

281 Haverhill Street.

MINIMAL SCARRING AFTER ELECTRODESICCATION

THOMAS S. SAUNDERS, M.D., PORTLAND, ORE.

For the most part, the electrodesiccating current is used for the removal of superficial rather than deep-seated growths and of benign rather than malignant lesions. Hence cosmetic considerations are of great importance.

The development of a hypertrophic scar after operation defeats the purpose (cosmetic improvement) for which the operation was attempted. Many practitioners feel that the formation of scars is a matter beyond their control, that the tendency for hypertrophic scars to develop represents an idiosyncrasy on the subject's part.

As a result of practical experience, I believe that the development of hypertrophic scars after electrodesiccation can be largely prevented (this does not apply to keloids, which are obvious idiosyncrasies). Frequent use of this modality has taught me that the formation of hypertrophic scars usually results from improper technic. Rapid, intense destruction of a lesion with a very hot spark seems to be almost always followed by a large, thick, elevated scar, particularly if the area is extensive. Conversely, the use of the smallest, "coldest" spark possible, with frequent interruption to allow the tissue to dissipate the heat, has almost always resulted in a flat scar of good cosmetic appearance. Such a technic is necessarily slow and time consuming, but the results justify the expenditures.

Medical Arts Building.

FAMILIAL LICHEN PLANUS

Report of Three Cases in One Family

MARVIN B. GOLDSTEIN, M.D., YOUNGSTOWN, OHIO

Saffron[1] reported 4 cases of lichen planus in one family and reviewed the literature to date. I have recently observed 3 cases in one family that I wish to report.

REPORT OF CASES

E. H., a white man aged 28, consulted me in November 1941 for an eruption on the legs of seven years' duration. The lesions continued to the anterior and lateral aspects of the tibias and were of the chronic hypertrophic variety of lichen planus.

Mrs. R. H., aged 47, mother of E. H., consulted me in February 1942 for an eruption which had been present for five years. She had hypertrophic lichen planus on both legs similar to her son's, and she also had lichen planus on the buccal surfaces of the cheeks.

Mrs. S. V., aged 19, sister of E. H., not living at the same address, consulted me in February 1942 for a generalized eruption which had been present for two and one-half months. She was being treated for poisoning from a fur coat. Pruritus in this case was intense, while in the other 2 cases it was negligible. Examination disclosed a typical acute widespread eruption of lichen planus. No oral lesions were noted in this case.

COMMENT

From these cases and from others reported in the literature, it appears that lichen planus is often familial, although this fact is seldom considered. Psoriasis is known to be present in several members of a family in many instances, and I believe that if this point is borne in mind lichen planus may be found to have a similar background.

404 Stambaugh Building.

---

1. Saffron, M. H.: Familial Lichen Planus, Arch. Dermat. & Syph. **42**:653 (Oct.) 1940.

Special Reports

FIRST MEETING OF THE CONSULTING STAFF OF THE DERMATOSES INVESTIGATIONS SECTION OF THE UNITED STATES PUBLIC HEALTH SERVICE*

The members of the consulting staff include: Drs. Paul A. O'Leary (chairman), Rochester, Minn.; S. William Becker, Chicago; Howard Fox, New York; Samuel M. Peck, New York; Richard L. Sutton Jr., Kansas City, Mo., and Harry J. Templeton, Oakland, Calif. Dr. O'Leary was unable to be present at this meeting, and Dr. Fox acted as temporary chairman. All other members of the consulting staff were present.

Much time was devoted to the wording of a definition for a primary cutaneous irritant and for a cutaneous sensitizer. The following definitions were finally selected:

A primary cutaneous irritant is an agent which will cause dermatitis by direct action on the normal skin at the site of contact if it is permitted to act in sufficient intensity or quantity for a sufficient length of time.

A cutaneous sensitizer is an agent which does not necessarily cause demonstrable cutaneous changes on first contact but may effect such specific changes in the skin that after five to seven days or more further contact on the same or other parts of the body will cause dermatitis.

There was discussion of the plan outlined by the United States Public Health Service for testing new fabrics, finishes, dyes and cosmetics to determine their possible skin irritating powers before they are sold to the public, as described in two recent articles (Schwartz, L., and others: An Outbreak of Dermatitis from New Resin Fabric Finishes, *J. A. M. A.* **115:** 906 [Sept. 14] 1940; Dermatitis from New Synthetic Resin Fabric Finishes, *J. Invest. Dermat.* **4:** 459-470 [Dec.] 1941). In the discussion of the plan it was brought out that it would be desirable to select the 200 subjects for patch tests at random from the employees of the factory rather than from the patients of a clinic or hospital, whose resistance might be lower than normal. The subject of insurance against suits for the physician performing such tests was discussed, and it was thought that the regular insurance carried by

* Held at the National Institute of Health, Bethesda, Md., April 20 and 21, 1942.

the physician would include this. The matter of fees to be paid for subjects submitting to patch tests was also discussed, and it was decided that this matter could be left to the individual physician.

Since occupational diseases of the skin constitute about two thirds of all industrial diseases, it was decided that in order to help keep workers on the job it would be advisable for the Public Health Service to give a course in occupational dermatoses to a number of representative dermatologists living in key sections of the country, who after taking the course might teach the subject to dermatologists and others concerned living in their locality. An announcement of the plan will be made in the ARCHIVES of DERMATOLOGY and SYPHILOLOGY and in the *Journal of Investigative Dermatology*. Dr. Schwartz will also submit the plan in his paper entitled "The Role of the Practicing Dermatologist in Our War Effort," which will be read before the Society for Investigative Dermatology in Atlantic City, N. J., in June. Members of the consulting staff will help select from the applications received dermatologists to whom the course will be given, taking into consideration the section of the country from which they come and the teaching qualifications of the applicant. The time, the place, the length of the course and other details will be decided later.

The work performed by the Dermatoses Investigations Section on poison ivy, war gases, protective clothing and protective ointments was reviewed.

Since dermatologists possess a special knowledge of the skin-irritating properties of chemicals, it was suggested that cooperation between the Dermatoses Investigations Section and chemists engaged in the manufacture of war gases might prove useful in developing new war gases and protective methods against them.

It was generally agreed that research for the near future by the Dermatoses Investigations Section should be in line with war efforts.

Desensitization against industrial cutaneous irritants was discussed, and it was agreed that desensitization experiments constitute research problems of the future, not only for the Dermatoses Investigations Section but for dermatologists in general. The best first method of approach toward desensitization was thought to be by gradual application of the substance to the skin either by bathing in dilute solutions, the strength of the bath to be increased as immunity develops, or by local treatment of parts of the skin, with similar increase in concentration.

Dr. Fox was requested to publish a notice in the ARCHIVES of DERMATOLOGY and SYPHILOLOGY stating that the Dermatoses Investiga-

tions Section is available for whatever services can be rendered to dermatologists in assisting them with their problems.

Official cards identifying the members of the consulting staff as consultants to the Dermatoses Investigations Section of the United States Public Health Service will be issued.

LOUIS SCHWARTZ, M.D.

NOTE.—At the conclusion of the meeting, the following resolution was unanimously adopted:

The consulting staff wishes to express its appreciation of the splendid work of Dr. Louis Schwartz in organizing and conducting the Bureau of Dermatoses Investigations. Mainly through his efforts, often based on original research, the practice of occupational diseases of the skin has been advanced to a high level.

News and Comment

EXAMINATIONS BY THE AMERICAN BOARD OF DERMATOLOGY AND SYPHILOLOGY

Written examinations will be held in various large cities in the United States on Monday, Oct. 12, 1942. The oral examination will be held in Chicago on Friday and Saturday, December 4 and 5. Applications must be received by the Secretary, Dr. C. Guy Lane, 416 Marlboro Street, Boston, by September 1.

Abstracts from Current Literature

EDITED BY DR. HERBERT RATTNER

PELLAGRA AND PORPHYRINURIA. ROBERT KARK and ARNOLD P. MEIKLEJOHN, Am. J. M. Sc. **201**:380 (March) 1941.

Chemical examinations by Kark and Meiklejohn suggest that porphyrinuria is seldom present in cases of pellagra unassociated with alcoholism, though a person with a history of alcoholism may present this symptom whether or not he has pellagrous dermatitis. They conclude that porphyrinuria is not an essential feature of pellagra and that when present it is perhaps consequent on some alteration of hepatic function.

THE TRIVALENT ARSENICALS IN SYPHILIS: PART II. TREATMENT REACTIONS. HERMAN BEERMAN and JOHN H. STOKES, Am. J. M. Sc. **202**:606 (Oct.) 1941.

Continuing their discussion of the trivalent arsenicals in syphilis, the authors review the reactions to the therapeutic use of these drugs. In fifteen pages they present their own impressions as well as the opinions of writers represented by a list of one hundred and eighty-six references. It is likely that any syphilologist will gain from a perusal of this work.

WERNER'S SYNDROME: REPORT OF THE FIRST NECROPSY AND OF FINDINGS IN A NEW CASE. B. S. OPPENHEIMER and VICTOR H. KUGEL, Am. J. M. Sc. **202**:629 (Nov.) 1941.

According to Oppenheimer and Kugel, Werner's syndrome is a familial disease characterized by premature graying and loss of hair, a scleroderma-like process and cataracts. The patient is usually short and slight, and there is likely to be evidence of hypogonadism. Three cases are described. There was tautness of the skin of the feet (in 1 case the hands were also involved), the skin was hard and shiny and ulceration was present. The authors suggest a possible relationship with hyperparathyroidism.

BIOLOGIC FALSE POSITIVE SEROLOGIC REACTIONS IN TESTS FOR SYPHILIS. CHARLES F. MOHR, JOSEPH E. MOORE and HARRY EAGLE, Arch. Int. Med. **68**:898 (Nov.) 1941.

The authors emphasize the necessity for recognition of falsely positive serologic reactions occurring in apparently normal persons and record 9 examples. Reagin or reagin-like factors have already been demonstrated in some normal human serums. The authors suggest that this normal factor may have been present above the threshold value in their cases.

LYNCH, St. Paul.

CUTANEOUS VASCULAR AND PIGMENTARY CHANGES IN CASTRATE AND EUNUCHOID MEN. EDWARD A. EDWARDS, JAMES B. HAMILTON, S. QUIMBY DUNTLEY and GILBERT HUBERT, Endocrinology **28**:119 (Jan.) 1941.

The cutaneous vascularity and pigmentation of 4 castrated males (including a Negro) and 5 eunuchoid patients were studied before, during and after treatment with testosterone propionate. Determinations were made with the aid of the Hardy recording spectrophotometer.

It is concluded that androgens have a profound influence on the vascularity and pigmentation of the entire human skin.

JACOBSEN, Buffalo. [AM. J. DIS. CHILD.]

NAIL LACQUER DERMATITIS. L. HOLLANDER, J. A. M. A. **115**:1714 (Nov. 16) 1940.

Hollander reports 3 instances of dermatitis due to sensitivity to nail lacquer. In all cases the eyelids and some other part of the face were involved. Since his experience showed that patch tests with suspected cosmetic contacts were inconclusive, a routine was developed in which cosmetics were all banned from immediate contact and later allowed to be used one by one. In all 3 instances the eruption returned within a few hours after the use of nail polish was resumed.

CHALK GOUT: A REPORT OF TWO CASES WITH A BRIEF SUMMARY OF SOME PREVIOUSLY REPORTED STUDIES ON CALCINOSIS. E. F. ROSENBERG, J. A. M. A. **115**:1791 (Nov. 23) 1940.

Rosenberg reports the cases of 2 patients with the localized form of calcinosis cutis. Both patients complained of pain and stiffness of the joints associated with symptoms of Raynaud's disease.

MILKERS' NODULES. F. T. BECKER, J. A. M. A. **115**:2140 (Dec. 21) 1940.

Becker in the first American paper on milkers' nodules, reports 4 instances of the disease. The lesions, which are located usually on the hands and fingers, consist of well demarcated papules which enlarge to form firm, reddened, semiglobular, umbilicated lesions. Involution occurs in from four to six weeks without scarring. The regional lymph nodes sometimes are enlarged, and rarely a generalized eruption occurs. The affected person invariably has been milking cows on whose udders open ulcers or crusted lesions were present. Becker believes the cause is a filtrable virus unrelated to vaccinia, since the reaction to a Paul test (keratitis following inoculation of a rabbit's cornea with material from lesions of smallpox or vaccinia) was negative in 2 of his cases in which it was tried. In 2 cases there was no immunity to smallpox vaccination. The clinical course, absence of scarring and histologic observations were other differential points.

MELANOMA OF THE CENTRAL NERVOUS SYSTEM: REPORT OF THIRTY-FOUR CASES, IN NINETEEN OF WHICH THE DIAGNOSIS WAS VERIFIED BY OPERATION OR NECROPSY. F. P. MOERSCH, J. G. LOVE and J. W. KERNOHAN, J. A. M. A. **115**:2148 (Dec. 21) 1940.

Moersch, Love and Kernohan report that in the decade from 1930 to 1939 they have observed approximately 500 cases of melanoma. In over 347 cases there was evidence of local recurrence or metastasis. In 34 of these, or 10 per cent, there was clinical evidence of melanotic involvement of the central nervous system. In 19 of these cases histologic confirmation was obtained at operation or necropsy; the correct diagnosis of metastasis to the central nervous system would have been overlooked in 12 cases without histologic evidence. In 8 of the 12 cases no primary lesion was discovered. The length of time from the excision of the primary melanoma to the death of the patient varied from four months to thirteen years; the average duration was approximately two and one-half years. In retrospect it was thought that most of the cutaneous lesions (primary melanomas) had been excised after they had increased rapidly in size. The authors believe that moles situated in places subject to irritation should be excised before they show any signs of "wildness."

MULTIPLE BENIGN CYSTIC EPITHELIOMA: REPORT OF TEN CASES IN ONE FAMILY. H. J. GOLDMAN, J. A. M. A. **115**:2253 (Dec. 28) 1940.

Goldman reports on the occurrence of multiple benign cystic epithelioma in 10 members of a family, the incidence being 60 per cent. The disease was transmitted at first from the male to the female, but subsequently it was passed through the female. There were 5 males and 5 females. While the distribution usually

included the sites frequently noted in textbooks, Goldman noted in 6 cases that the region in front of the ear was involved. Twelve biopsies were carried out in 7 cases. The presence of keratinous nests, thought to be due to dilatation of the hair follicle, was the most consistently present, most conspicuous and most easily identified pathologic change in the entire series. The presence of the nests was considered important in two specimens which would otherwise have been considered examples of basal cell epithelioma.

DERMATITIS DUE TO A FACE POWDER CONVEYOR. L. HOLLANDER, J. A. M. A. **115**:2271 (Dec. 28) 1940.

Hollander reports an instance of dermatitis of the face due to sensitivity to a rubber sponge powder puff.

LONG-TERM RESULTS IN THE TREATMENT OF EARLY SYPHILIS. P. PADGET, J. A. M. A. **116**:7 (Jan. 4) 1941.

Padget evaluates the long term results of treatment of early syphilis from a consideration of the status of 551 patients who had been completely reexamined five or more years after termination of the original treatment. Two hundred and seventy-three of these patients were followed for more than ten years. Two thirds of the patients were considered cured. Those who were apparently well at five years remained so at later periods. Padget concluded that the alternating continuous treatment system was superior to other methods and that the result is favorably affected by the amount of arsenic given in the early months.

A CLINICAL AND PATHOLOGIC STUDY OF ORAL DISEASE: BASED ON 2,300 CONSECUTIVE CASES. F. P. MCCARTHY, J. A. M. A. **116**:16 (Jan. 4) 1941.

According to McCarthy, the recognition and treatment of diseases of the oral cavity is an important yet neglected field of medicine. His report is based on 2,300 consecutive cases and includes a discussion of various disorders under the headings of gingival lesions, stomatitis and lesions of the tongue and of the lip. He further discusses the subject under the headings of neoplasms and cysts, dermatoses, blood dyscrasias and constitutional diseases, drug eruptions, leukoplakia and special and miscellaneous lesions. McCarthy found leukoplakia to be the most frequent and most important lesion of the oral cavity (322 cases, or 14 per cent). The cause and treatment of gingival lesions were found to be unsatisfactory. He reports 26 cases of oral lichen planus without cutaneous lesions. He does not recognize an entity described as following an electrolytic burn, believing that lesions attributed to this factor can be explained in other ways. He also draws attention to the frequent occurrence of hyperplasia of the gums in persons with epilepsy given dilantin sodium therapeutically.

CHROMOBLASTOMYCOSIS: REPORT OF THE SIXTH CASE FROM CONTINENTAL UNITED STATES. C. W. EMMONS, H. HAILEY and H. HAILEY, J. A. M. A. **116**:25 (Jan. 4) 1941.

This article briefly describes the previously reported cases of chromoblastomycosis and gives the essential features of the sixth case to be reported from the United States. The patient was a farmer aged 68 who had a verrucous lesion on the left wrist for three months. The appearance was typical for blastomycosis. The mycologic examination revealed Hormodendron pedrosoi. Treatment included intensive potassium iodide therapy together with two mild exposures of roentgen rays and resulted in complete disappearance of the lesion after three months. The authors believe the name of the disease (chromoblastomycosis) should be retained and that while the etiologic agents of the disease (H. pedrosoi and Phialophora verrucosa) are probably related, it is unwise to change either name at present, as such a change would probably only be temporary.

ADRENAL CORTEX EXTRACT IN THE TREATMENT OF BROMIDE ERUPTION AND BROMIDE INTOXICATION. C. P. BONDURANT and C. CAMPBELL, J. A. M. A. **116**:100 (Jan. 11) 1941.

Bondurant and Campbell report that the administration of solution of adrenal cortex in combination with sodium chloride is more effective than sodium chloride alone in the treatment of bromide intoxication and of bromoderma. Of 9 patients with bromide intoxication, 7 received the combined therapy, and mental symptoms disappeared in an average of five and one-half days. The other 2 received sodium chloride alone, and their symptoms lasted an average of fifteen days. Seven patients in a hospital for persons with mental disease were given bromides until the blood values for bromide reached toxic levels. Four of these received the combined therapy while 3 received sodium chloride alone. The results obtained confirmed the results of the first series.

CHEILITIS FROM SENSITIVITY TO OIL OF CINNAMON PRESENT IN BUBBLE GUM. J. MILLER, J. A. M. A. **116**:131 (Jan. 11) 1941.

A woman aged 25 had cheilitis for six months. There were no lesions elsewhere on the body. Oil of cinnamon used by the firm for which she worked was suspected as the cause because of a positive reaction to the patch test. Since there was no direct contact in her work Miller employed a fume test, which showed that the vapor alone was capable of initiating a reaction. Care to avoid close proximity to oil of cinnamon brought about an apparent permanent cure.

BLASTOMYCOSIS AND THE BLASTOMYCOSIS-LIKE INFECTIONS. L. M. SMITH, J. A. M. A. **116**:200 (Jan. 18) 1941.

Smith compares and differentiates the various rare mycoses which might be confused with Gilchrist's blastomycosis. He also describes the characteristics of the causative fungi, charting the cultural and microscopic features by which they may be recognized. He stresses the value of a laboratory diagnosis as well as of an awareness of the possibility of the diagnosis of a fungous disease. In 5 cases of coccidioidal granuloma and in 1 case of sporotrichosis there were a positive reaction to antigen made from the specific micro-organism and negative reactions to control vaccines. Smith believes that in both of these diseases as well as in blastomycosis, the use of sensitizing antigens may be an important adjunct to other methods of diagnosis.

SULFANILAMIDE IN THE THERAPY OF ACTINOMYCOSIS. L. DOBSON, E. HOLMAN and W. CUTTING, J. A. M. A. **116**:272 (Jan. 25) 1941.

Dobson, Holman and Cutting obtained favorable effects in 3 patients with actinomycosis by administering a combined therapy of sulfanilamide, iodides and roentgen rays. They believe that while the effect of sulfanilamide was partially because of its action on secondary invading micro-organisms, the rapid response suggested a direct effect as well.

THE USE OF BISMUTH COMPOUNDS IN SYPHILOTHERAPY. E. N. WALSH and S. W. BECKER, J. A. M. A. **116**:484 (Feb. 8) 1941.

Walsh and Becker record the results of treatment of 55 patients with early syphilis and compare these results with those obtained by the Cooperative Clinical Group. Whereas the plan of procedure of the group entailed continuous treatment with alternating courses of an arsenical and a bismuth compound, the patients of Walsh and Becker received the arsenical combined with a bismuth compound, and bismuth therapy was continuous for the first forty-two weeks. Thereafter bismuth was given in courses interspersed with rest periods. The total number of injections of the bismuth compound advocated was eighty-three, compared with thirty-eight in the scheme of the Cooperative Clinical Group. The results recorded

by Walsh and Becker for this scheme of treatment showed 100 per cent satisfactory results, as compared with 79.7 per cent by the group. The authors also found that prolonged bismuth therapy in place of fever and tryparsamide treatment gave satisfactory results in patients with asymptomatic neurosyphilis and tabes dorsalis.

LEWIS, New York.

THE TREATMENT OF ACUTE STOMATITIS. CHARLES HENDEE SMITH and HERBERT B. JOHNSON, J. Pediat. **17**:1 (July) 1940.

This paper discusses the treatment of acute gingivostomatitis. So many remedies have been recommended that it is probable that most of them are worthless. The authors present a technic for treatment which has been highly satisfactory. The mouth is cleansed with a weak solution of hydrogen peroxide to remove accumulated sordes or sloughing material, and this is followed by a mouth wash, compound solution of sodium borate N. F. (Dobell's solution) or a solution of sodium perborate being used. The gums and ulcers are then dried carefully with several swabs of cotton. Then a 5 to 7 per cent solution of chromic acid is applied by cotton swab directly to the ulcers and the inflamed gums. This treatment is repeated twice daily for one to three days and then once daily. Between treatments the patient may use a mouth wash. Supportive treatment such as is used for any febrile illness must be given. The chromic acid has a definite analgesic effect, and the patients are able to eat with more comfort for several hours after treatment. There is rapid improvement in the inflamed gums, and the course of the disease is definitely shortened, especially in those cases in which treatment is begun early.

RAUH, Cincinnati. [AM. J. DIS. CHILD.]

THE INFLUENCE OF VITAMIN C ON THE THERAPEUTIC ACTIVITY OF BISMUTH, ANTIMONY AND THE ARSENIC GROUP OF METALS. SIMON L. RUSKIN and M. SILBERSTEIN, M. Rec. **153**:327 (May 7) 1941.

The oxidation of heavy metals plays an essential role in their chemotherapeutic activity. Vitamin C is one of the predominating oxidation-reduction mechanisms in the body. Therefore, the authors suggest the use of ascorbic acid, not alone as a detoxifying agent but as part of the molecular compound embodying the therapeutic metal. Employing a preparation of bismuth cevitamate (ascorbate), a bismuthyl derivative of the sodium salt of ascorbic acid, they obtained excellent results in 14 cases of Wassermann-fast syphilis. Only 1 patient responded unfavorably to the drug; each injection was followed by nausea, chills and debility. On the basis of comparative studies, it is claimed for bismuth cevitamate (ascorbate) that its therapeutic index is substantially higher than that of the commonly used preparations of bismuth. The authors report only on the use of a bismuth preparation, but the same principles could be applied to therapy with arsenic and antimony.

RATTNER, Chicago.

RIBOFLAVIN DEFICIENCY: REPORT OF A CASE IN A CHILD WITH CURE BY SPECIFIC TREATMENT. WILLIAM P. SHIELDS, New England J. Med. **223**:215 (Aug. 8) 1941.

Riboflavin deficiency is often evidenced by cheilosis. In cases of this deficiency there are often manifestations of other deficiency diseases, such as pellagra, scurvy and polyneuritis.

A 6 year old girl of Italian descent had been born prematurely and had had no cod liver oil or orange juice since birth. The diet of the family was restricted because of a limited income, $6 a week being expended for food for a family of 6. The patient was the only member of the family in whom specific lesions developed. The lesions were ulcerating and involved the upper and lower lips, and there were deep transverse fissures at the angles of the mouth, not including the buccal mucosa.

Riboflavin was given in doses of 15 mg. daily for three days. Two days from the beginning of its administration there was almost complete resolution, and

10 mg. was then given for three days. But three days after administration of the drug was stopped the lips showed signs of a relapse. Administration of the riboflavin was then continued till recovery ensued.

ECZEMA IN INFANTS AND YOUNG CHILDREN. LEWIS WEBB HILL, New England J. Med. **223**:624 (Oct. 17) 1940.

Eczema in infants and young children may be caused by allergy to foods, to inhalants and to organic or inorganic contactants, by fungous or low grade pyogenic infection and by chemical or mechanical irritation. However, in most cases it is due to allergy.

Typical allergic eczema, or "atopic dermatitis," seldom begins before the age of 3 months. The diagnosis of atopic dermatitis in an infant is suggested by the character of the eruption and the severe itching and may often be confirmed by positive results of cutaneous tests. A positive reaction to a scratch test is usually of etiologic significance; a positive reaction to an intracutaneous test may or may not be.

A combination of immunologic and local treatment is much better than either alone. Many infants are relieved of their dermatitis when certain foods, notably milk and wheat, are removed from the diet.

In dealing with sensitivity to milk in eczema, goat's milk or a milk-free food may be of value. Milk-free foods, most of which are made of soybean flour, sometimes cause bulky, loose bowel movements and irritation of the buttocks. If the formula is cooked in a double boiler for three quarters of an hour, this tendency is considerably lessened.

Wheat is the most allergenic of the cereals. Oranges frequently cause reactions; even when cutaneous reactions to orange are negative, it is not uncommon for itching to be severer after taking it. Of the vegetables, peas and spinach are the most allergenic. Sensitivity to feathers is not uncommon, and silk and kapok are occasionally causes of dermatitis.

Face masks are not recommended. A rectangle of cellophane taped to the part of the bed sheet underneath the head is valuable in preventing trauma due to rubbing. Neither lotions containing insoluble powders nor salves are desirable, as a rule, during the acute, weeping stage. Wet compresses of 2 per cent resorcinol or tannic acid solution containing 2 per cent boric acid, 1 per cent aluminum acetate solution or 0.05 per cent potassium permanganate solution are better. Some of the white tar preparations on the market are fairly satisfactory, but none is as good as coal tar ointment. The latter should not be employed, however, if pyogenic infection is present.

House dust causes dermatitis more often by inhalation than by outside contact.

GENGENBACH, Denver. [AM. J. DIS. CHILD.]

STUDY OF THE SERUM LIPIDS IN SCLEREMA NEONATORUM. LUIGI LUZZATTI and ARILD E. HANSEN, Proc. Soc. Exper. Biol. & Med. **46**:325 (Feb.) 1941.

Study of the subcutaneous fat tissue and of the serum lipids of a 3½ month old, breast-fed girl with sclerema neonatorum was made, both at the time the disease was manifest and when all evidence of the disorder had disappeared. It was believed that this approach to the problem might help to provide a better understanding of the nature of the disturbance and might also help in detecting any evidence of a metabolic abnormality which may be associated with this condition. The histologic appearance and the chemical structure of this tissue revealed the known alterations: chronic inflammatory reaction and presence of abnormal amounts of saturated fatty acids (iodine number, 42). When the condition was improved, repeated analyses revealed normal adipose tissue, both histologically and chemically (iodine number, 57). Early in the disease an increase in all the fractions of the serum lipids was present; associated with this was a decrease in the iodine number, especially of the neutral fat. When all signs of the disease had disappeared, the blood lipids were normal. Fat absorption as shown by balance studies and by blood studies was within normal limits. The lipemia is

discussed briefly in the light of the alterations of the fat tissue, and it is suggested that a disturbance in the deposition of fat or in its transportation from the pathologic areas may be present in this disease. The clinical observation gives support to the idea that this tissue was metabolically altered.

HANSEN, Minneapolis. [AM. J. DIS. CHILD.]

ROENTGEN THERAPY OF HEMANGIOMATA. FRED M. HODGES, L. O. SNEAD and R. A. BERGER, Radiology **36**:700 (June) 1941.

The authors report on the treatment of 19 infants with angioma by means of contact roentgen therapy. The results were uniformly good. In three seconds 400 r measured in air was delivered to the affected areas every four to six weeks. In the discussion one of the authors agreed that if the dose is reduced to 200 r, there is less likelihood of telangiectasia. In some cases the rays were filtered. This method is rapid, safe and efficacious.

REVIEWER'S NOTE.—Radium plaques will probably be used less in the future by dermatologists and others, especially since shock-proof apparatus can be used close to the skin with no danger to the patient or to the parent or assistant holding the infant. Also a large dose can be delivered in a few seconds.

CIPOLLARO, New York.

CONGENITAL SYPHILIS IN ONLY ONE OF TWINS. F. R. SMITH JR. and J. M. SPENCE JR., South. M. J. **34**:147 (Feb.) 1941.

The authors cite 12 previously reported cases of congenital syphilis occurring in only one of twins. Of these 5 were completely acceptable; the others, while probably authentic, suffered from lack of documentation of important detail. They present the criteria which must be fulfilled before the diagnosis of congenital syphilis in only one of twins may be made.

The authors then report 5 instances, from their own practice and from the records of Johns Hopkins Hospital, in which congenital syphilis occurred in only one of twins. They point out that since 4 of these 5 instances were observed in 44 twin pregnancies to 40 women with syphilis, the condition is not so rare as might be expected.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

NEURONITIS PRODUCED BY THALLIUM ACETATE POISONING AND CURED WITH CONVULSANT DOSES OF METRAZOL. CARLOS F. KRUMDIECK, Rev. d. Hosp. d. Niño **1**:5 (March) 1940.

The author records the case of a 9 year old girl poisoned with thallium acetate while being treated for tinea tonsurans. Cerebellar and mental symptoms demonstrated the action of the drug on the neurons of the cortex and the cerebellum. Because all of the common treatments for this type of poisoning failed and because he had employed metrazol before in treating similar syndromes referable to the nervous system, he used huge doses of the drug in this case, until convulsions appeared. A dose of 4 cc. was administered intravenously every four days, and four treatments in all were given. The results were splendid.

He comments on the dangers of using thallium acetate and recommends careful study of the patient from the neurologic point of view before it is used. He also urges the treatment of other patients with toxic neuronitis of this type with metrazol, so that his results may be confirmed or rectified.

JESÚS LOZOYA, Mexico City, Mexico. [AM. J. DIS. CHILD.]

PRELIMINARY REPORT ON THE TREATMENT OF SCABIES WITH BENZYL BENZOATE. LUIS NÁJERA ANGULO and BERNABÉ JIMÉNEZ ROLDÁN, Rev. méd. latinoam. **26**:793 (April) 1941.

A single application of benzyl benzoate lotion cured 52 patients suffering from scabies, and two applications cured 28 patients. There were no failures. The authors found the treatment well tolerated even by women and children, without

the production of dermatitis or secondary eczematizations. Complicated scabies was not aggravated; in fact, it was most often favorably influenced. It was not necessary for the patient to change his clothing, and only a preliminary cleansing hot bath was used.

LEIFER, New York.

HERPES ZOSTER ACCOMPANYING MENINGITIS IN INFANCY. ISTVÁN BRÜNDLMEIER, Orvosi hetil. **84**:259 (May 18) 1940.

A 9 month old infant with the symptoms of meningitis was observed. The infant also had bilateral otitis media and on the left side a patch of herpes zoster a handbreadth wide, stretching forward on the trunk from the spine. The cerebrospinal fluid was water clear, but the Pandy reaction was positive and there were 200 lymphocytes per cubic millimeter. The occurrence of herpes zoster with meningitis is not very rare, but it has not yet been observed in infancy. The infant recovered completely in two weeks.

GÖTTCHE, Budapest, Hungary. [AM. J. DIS. CHILD.].

THREE CASES OF NODULATE IRITIS (IRITIS BOECK?). P. KINDT, Acta ophth. **18**:38, 1940.

The author describes 3 cases of sluggish iritis occurring in adult women and characterized by the formation of nodules, little pain and a fair tendency toward healing. Provisionally he regards them as being cases of Boeck's sarcoid. The nodules were grayish and semitransparent. They were fairly large, many being visible to the unaided eye. The smaller ones were smooth and round, whereas the larger ones were slightly uneven and somewhat irregular in shape. A few were so prominent as to protrude into the anterior chamber. They showed no tendency to grow into the sclera. They were interlaced and covered on the surface by fine vessels and thus were sharply outlined from the contiguous iris tissue, which appeared normal and without vascularization. Tuberculous nodules are not so sharply outlined, merging gradually into iris tissue which is often vascularized.

UVEOPAROTID FEVER IN A CASE OF BOECK'S SARCOID. E. DALSGAARD-NIELSON, Acta ophth. **18**:210, 1940.

A man of 28 had swelling of both parotid glands, together with bilateral iridocyclitis. Roentgenograms of the lungs showed a considerable enlargement of the hilar shadows on each side, but the bones of the hands and feet showed no characteristic changes. A piece of the parotid gland was excised and revealed changes which were interpreted as those of Boeck's sarcoid. An iridectomy was performed on one eye because of secondary glaucoma, but the iris tissue, which had failed to show nodules clinically, revealed no microscopic changes characteristic of sarcoid. The author believes that the case demonstrates a close relation between uveoparotid fever and benign lymphogranulomatosis.

MOLLUSCUM CONTAGIOSUM CORNEAE. R. SYSI, Acta ophth. **19**:25, 1941.

A 3 year old child presented the typical nodular lesions of molluscum contagiosum on the skin of the cheek, forehead, upper lip and border of the left upper eyelid.

The left eye showed a slight pericorneal injection and a central area of corneal infiltration about 4 mm. in diameter. In the midst of this infiltration was an elevated nodule about 1.5 mm. in diameter with an umbilicated center. This was excised, together with the nodule on the eyelid. The nodule from the skin showed the typical picture of molluscum contagiosum; that from the cornea showed pigmented corneal cells suggestive of cells of molluscum contagiosum, but the small size of the specimen made a satisfactory histologic examination impossible.

O. P. PERKINS. [ARCH. OPHTH.]

DIABETES INSIPIDUS CAUSED BY BOECK'S SARCOID. M. JERSILD, Nord. med. (Hospitalstid.) **1**:743 (March 18) 1939.

A 15 year old boy who had been suffering from diabetes insipidus for twelve months began to have various signs of Boeck's sarcoid, namely, roentgen changes in the lungs, iridocyclitis, swelling of the parotid gland and universal adenitis. Furthermore, there was a cutaneous disturbance, and biopsy of the skin revealed Boeck's sarcoid. The reaction to a Mantoux test with 1 mg. of old tuberculin was negative. Presumably, the diabetes insipidus was caused by involvement of the hypophysis by the sarcoid process. The Swede Tillgren and the Frenchman Lesné have reported similar cases.

WALLGREN, Göteborg, Sweden. [AM. J. DIS. CHILD.]

Society Transactions

MANHATTAN DERMATOLOGIC SOCIETY

DAVID L. SATENSTEIN, M.D., *President*
ANTHONY C. CIPOLLARO, M.D., *Secretary*

Jan. 13, 1942

Ulcus Vulvae Acutum. Presented by DR. GEORGE C. ANDREWS.

R. M., a woman aged 21, a bookkeeper, presents recurrent ulcers of the vulva. The lesions which the patient had about one year ago were much larger and more numerous. At that time the ulcers had been present for eight months and had not been affected by various methods of treatment. At different times there have been small superficial ulcers on the lips and buccal mucosa which resembled aphthous ulcers. The oral lesions healed spontaneously but recurred.

The present ulcers appeared about two weeks ago. The lesion on the right labium majus is about 1 inch (2.5 cm.) in diameter and is a rounded, punched-out ulcer with rather smooth walls. The base is a granulomatous, firm mass covered with a greenish seropurulent exudate. There is some inflammatory reaction about the ulcers. They have been rather painful. There is a similar but smaller ulcer on the left side of the vulva, and several scars can be seen from previous attacks about one year ago.

Dark field examinations, several Wassermann tests of the blood and the Frei test gave negative results. The Ito-Reenstierna test elicited an area of erythema which measured 6 by 7 cm., with some central induration. Microscopic sections on two different occasions showed only nonspecific granulation tissue; no Donovan bodies were found. On several occasions smears from the lesions have shown long thick gram-positive bacilli, presumably Bacillus crassus.

During the first attack, one year ago, the ulcers healed rather quickly with the administration of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) internally, rest in bed and application of potassium permanganate locally.

Pseudoxanthoma Elasticum. Presented by DR. GEORGE C. ANDREWS.

M. F., a woman aged 19, a stenographer, presents an eruption involving the neck, shoulders and upper parts of the chest and back. According to the history, the eruption has been present since her early childhood and has shown little change since then.

The individual lesions are small, circumscribed, yellow or cream-colored firm papules and nodules. Some of them tend to have a rather rectangular shape and vary in size from that of a pinhead to that of a large pea. While some of the lesions are discrete, others tend to coalesce and form irregular patches. The upper part of the chest shows numerous small white atrophic spots. There seems to be some loss of elasticity in the affected areas. The patient has also a mild seborrheic dermatitis of the scalp and acne vulgaris, with a few comedos and occasional papules on the face. There are many small pitted scars on the face, from previous acne lesions.

There is no similar disease of the skin in any member of her family.

Examination of the eyegrounds showed angioid streaks. A histologic examination, made about two years ago at the New York Hospital, confirmed the clinical diagnosis.

DISCUSSION

DR. ISADORE ROSEN: I disagree with the diagnosis of pseudoxanthoma elasticum. The lesions have a yellowish tinge and may be related to xanthoma.

DR. GEORGE M. LEWIS: Coarse wrinkling due to loss of elasticity in the skin is a cardinal sign of pseudoxanthoma elasticum and is not seen in this patient.

DR. PAUL E. BECHET: I agree with the diagnosis, which, furthermore, is proved by the histologic report.

DR. E. WILLIAM ABRAMOWITZ: I think the most probable diagnosis is syringocystadenoma.

DR. DAVID BLOOM: In the cases of pseudoxanthoma elasticum which I have observed the lesions were located on the neck and in the axillas and consisted of streaks and not of circumscribed small lesions such as this patient presents.

DR. FRED WISE: I believe the eruption is syringocystadenoma.

Hereditary Hemorrhagic Telangiectasia. Presented by DR. DAVID BLOOM.

B. M., a girl aged 7 years, of Irish descent, came to the New York Skin and Cancer Hospital on Dec. 19, 1941 with an eruption on the face, hands and forearms which had been present since the age of 3 years. The patient has spontaneous nosebleeds about once in a fortnight, which are moderate in intensity and stop quickly. Her birth was normal, and she had a normal infancy and childhood.

General examination showed nothing abnormal except for the cutaneous lesions. On the cheeks, the cutaneous portion of the lips and the backs of the hands and forearms there are numerous red puncta and linear or semicircular red streaks. The mucous membranes of the mouth, nose and throat are normal.

A paternal cousin, a boy, has a similar eruption.

Examination of the blood showed 4,050,000 erythrocytes, 83 per cent hemoglobin (13.9 Gm. per hundred cubic centimeters), 12,000 leukocytes and a normal differential count. There were 240,000 platelets; the bleeding time was seven and one-half minutes; the coagulation time was twenty-three minutes (capillary method), and the prothrombin time was sixteen seconds.

Hereditary Hemorrhagic Telangiectasia. Presented by DR. DAVID BLOOM.

M. M., a girl aged 12 years, the sister of the previous patient, shows an extensive involvement of the face, lips, neck, arms and forearms and a slight eruption on the palms.

The eruption consists of red puncta and red tiny streaks and circles. Below the chin and extending down to the neck there is an irregularly shaped plaque of naevus flammeus. A similar plaque is seen on the anterior and inner aspects of the right arm. The mucous membranes of the mouth are free except for some lesions on the inner aspects of the lips.

The eruption has been present since birth. The patient has had mild epistaxis about once a month.

DISCUSSION OF TWO CASES OF HEREDITARY HEMORRHAGIC TELANGIECTASIA

DR. DAVID BLOOM: The presence of naevus flammeus in one of the sisters is of great interest to me. The inherited predisposition to telangiectasia is probably the underlying factor in the formation of the nevus.

Keratolysis Exfoliativa. Presented by DR. GEORGE C. ANDREWS.

H. L. W., a man aged 27, an employee in a department store (drapery goods), recently consulted me because of an eruption on the palms and soles. Examination at this time showed a maculopapular eruption with a rather uniform distribution on the palms. The general health has been good. There is no history of past illness except for measles and whooping cough in early childhood. There is no personal history of ingestion of drugs or of allergy. His mother and sister have hay fever. He has never had any other disease of the skin, and there is no one in his family with a similar type of disease. The tonsils have been removed, and there is no evidence of any foci of infection.

The individual lesions vary in size from that of a pinpoint to that of a pinhead and seem to be rather deeply situated in the skin. The older lesions are dry and leave a small area of dry exfoliation, which is only slightly larger than the early papules. These lesions remain discrete. The soles show a similar but less extensive type of eruption. The face shows a discrete, erythematous, papular eruption on the cheeks, with a peculiar yellowish discoloration in the infraorbital regions. No vesicles or pustules are present, and, according to the patient, there have never been any. The extremities are cool and show a moderate hyperhidrosis. The disease has been present for ten years. It has varied slightly in severity, but the only period of freedom followed a series of roentgen ray treatments and lasted three or four months.

Cultures of material from the lesions have been negative for both bacteria and fungi. The basal metabolic rate was —14 per cent. The blood count and the urine were normal.

Many different kinds of topical drugs have been used without benefit.

DISCUSSION

Dr. Maurice J. Costello: This patient has pompholyx of the hands and feet, and the lesions on the face have a somewhat similar appearance.

Dr. Paul E. Bechet: The term keratolysis exfoliativa, first proposed by Carayon in 1903 (*Ann. de dermat. et syph.* **4**:283, 1903) and later used by Wende (*J. Cutan. Dis.* **37**:174, 1919), should be used with caution, as MacKee and Lewis (Arch. Dermat. & Syph. **23**:445 [March] 1931) stated that the condition is a dermatophytid, an opinion also expressed by Finnerud and Osborne in their discussion of the paper by MacKee and Lewis. Microsporon lanosum was cultivated in a case of keratosis exfoliativa in 1921 by Arthur W. Greenwood. The fact that examinations did not reveal fungi does not disprove that these lesions are dermatophytids.

Dr. Samuel M. Peck: It has been my impression that the diagnosis of ids is being made much too freely. It is agreed that it is sometimes difficult to differentiate a vesicular dermatophytid from a rather rare condition, such as a true dyshidrosis. This patient, however, seems to me to have the classic changes following hyperhidrosis with a resulting maceration of the skin. Perspiration on the palms and soles is not affected by changes in temperature but is easily influenced by mental effort. It is therefore seen in highly nervous patients. Also, there is continuous insensible perspiration on the palms and soles. If the perspiration is profuse, it is practically all water, and a condition which resembles the cutaneous changes after prolonged contact with water is seen.

Dr. George M. Lewis: At present the eruption has the clinical characteristics of pompholyx rather than of keratolysis exfoliativa. In the latter condition vesiculation is not present.

Dr. George C. Andrews: I believe that this boy has a congenital defect of the skin of the face. The changes are not due to roentgen ray treatment because they were present before he received such treatment. The eruption on the palms and soles is pompholyx. I am sure that no fungi are present.

Anetoderma of Jadassohn. Presented by Dr. Fred Wise.

H. P., a woman aged 26, registered at the New York Skin and Cancer Hospital on Sept. 30, 1939 with an eruption of three years' duration. The family history is irrelevant. She is subject to supraorbital headaches about twice a week, which last for from several hours to a whole day. These are sometimes associated with nausea and are occasionally severe. Her general health has otherwise been good.

The initial lesion started five years ago on the outer surface of the middle of the left arm as a "nail-sized red blotch," which remained as such for one year. Similar-sized blotches appeared a year later, one on the left arm above the first

lesion and one in the corresponding location on the right arm. These lesions never regressed but kept on enlarging slowly for three years. Last year lesions appeared on the back and six months ago on the scalp. During the past year the patient observed that every few weeks half of a lesion would become elevated for about three hours and become hard, white and itchy, while the other half would become redder. A margin of about ⅛ inch (0.32 cm.) around the entire lesion would also become red.

There are two lesions on the outer surface of the left arm, one on the right arm, two in the right scapular region which corresponds to the outline of the strap of the brassiere and two on the scalp. They are purplish to purplish red and well defined and vary in size from 1 to 3 by 4 cm. There are two ill defined, dime-sized erythematous lesions on the frontal and lateral regions of the scalp. The growths on the arms are raised 2 to 4 mm.; their surfaces are somewhat irregular, and the skin is thinned, finely wrinkled and atrophic. They show a slight depression on palpation. They are easily compressible and disappear almost entirely on diascopic pressure, leaving a thin erythematous area with fine telangiectatic capillaries. There is no difference in sensation between the lesions and the adjacent skin.

The results of routine laboratory tests were negative. On Jan. 6, 1942 intradermal tuberculin tests elicited a 1 plus reaction to a 1:100,000 dilution and a 2 plus reaction to a 1:10,000 dilution. Roentgenograms of the chest and skull were normal except for showing "slight membranous infiltration and hypertrophic thickening of both antrums and the right ethmoid."

A histologic section taken from the lower lesion on the left arm on Sept. 30, 1939 was diagnosed by Dr. D. L. Satenstein as "toxic dermatitis, with no evidence of sarcoid." It was described as follows: "The blood vessels throughout the whole section are dilated and surrounded by a cellular infiltrate composed of round cells and wandering connective tissue cells. The collagen bundles show parenchymatous edema throughout. The epidermis is thin and flattened. It shows no other changes." Another section, taken from the upper nodule of the left arm on Dec. 30, 1941, was diagnosed by Dr. Charles F. Sims as conforming to anetoderma of Schweninger and Buzzi. The description was as follows: "The epidermis presents no noteworthy changes. The cutis shows a moderate interstitial edema of the collagen. The blood vessels are moderately dilated, their walls somewhat edematous. There is a sparse nonspecific cellular infiltration surrounding the vessels in the upper part of the cutis. The elastic tissue is absent in most of the section, and where it is present it is sparse and fragmented."

DISCUSSION

DR. PAUL E. BECHET: I agree with the diagnosis of multiple benign tumor-like new growths, despite the fact that some of the characteristic clinical symptoms are lacking. The resemblance to sarcoid of Boeck is strong. This is especially true of the lesions on the back. On the arms, however, the skin overlying the small tumor-like growths is thinned, wrinkled and atrophic, and the characteristic hernia-like feel on palpation is easily demonstrated.

Folliculitis Decalvans Associated with Lichen Spinulosus. Presented by DR. E. WILLIAM ABRAMOWITZ.

M. M., a woman aged 33, registered at the New York Skin and Cancer Hospital on Dec. 19, 1941 with an eruption on the scalp and body of six months' duration. According to the history obtained from the patient and her attending physician, the eruption first appeared on the scalp after exposure to the sun. It began with pruritus of the scalp, and this was followed by lumps, pustules and alopecia in different areas. The lesions on the trunk were at first erythematous and pruritic, resembling prickly heat. Later they became rough with a nutmeg-grater-like feel.

The patient now presents a diffuse, mild erythema with plugged follicles. The eruption involves the trunk and upper extremities and extends down to the thighs. The scalp presents numerous areas of alopecia which are erythematous and show small numbers of pustules at the periphery of the plaques. Some of the lesions in the occipital region are fluctuating.

One month ago the attending physician administered sulfathiazole (2-[para-aminobenzenesulfonamido]-thiazole), 2 Gm. (30 grains) daily, for four days; this course was repeated later. The patient responded beneficially to this treatment, for the numerous pustules of the scalp disappeared rapidly. However, the lesions on the trunk were not influenced. A few days ago sulfathiazole was administered again, and after a dose of 1 Gm. the patient had a high fever, chills and headache associated with edema of the tongue.

Laboratory examination revealed no dyscrasia of the blood, and the urine was normal. There was a normal amount of total carotenoids in the blood (50.5 units of total carotenoids per hundred cubic centimeters and 15 blue units per hundred cubic centimeters). The basal metabolic rate was normal. Biopsy of skin from the left shoulder and from the scalp was reported as showing lichen planus pilaris.

DISCUSSION

DR. PAUL E. BECHET: The lesions on the scalp consist of a number of grouped, various-sized, tumor-like lesions, painful on pressure, with evidence of fluctuation in a few. There are extensive scarring and an irregular alopecia, which is entirely different from that following folliculitis decalvans. It is my impression that the lesions of the scalp represent an early stage of perifolliculitis capitis abscedens et suffodiens.

DR. DAVID BLOOM: The numerous follicular pustules on the scalp and the fluctuating boggy masses in the occipital region together with the lichen spinulosus eruption on the body force one to consider the eruption as a toxic manifestation, as a bacterid. The history offers another point of interest, namely, sensitization to sulfathiazole. Four weeks ago she received 2 Gm. daily of sulfathiazole for four days without any by-effects. A few days ago, however, after one dose of 1 Gm. of sulfathiazole, alarming symptoms developed—edema of the glottis and tongue, high fever, chills and rigidity of the neck. This case illustrates the necessity of caution in the administration of this drug.

DR. ISADORE ROSEN: The clinical features in this case do not coincide with those of folliculitis decalvans. The manifestations are too extensive and destructive. There is no question but what the patient is suffering from a generalized toxic eruption of the type ordinarily classified as an id. While the lesions on the trunk are spinulous, they do not suggest lichen spinulosus.

DR. SAMUEL M. PECK: I agree with Dr. Rosen, that the condition is some infectious process of the scalp which gives rise to "ids" on the shoulders and back analogous to lichen trichophyticus, which is often seen associated with kerion celsi. Histologically it may resemble lichen planus, plus a pronounced follicular hyperkeratosis. Clinically I do not see any other evidence for a diagnosis of a vitamin A deficiency. Such lesions as those on the back are known to occur as "ids," secondary to a fungous infection, and as a drug eruption, especially due to arsenic.

A Case for Diagnosis (Pili Torti?). Presented by DR. FRED WISE.

R. Z., a white girl aged 12 years, registered at the New York Skin and Cancer Hospital on Dec. 27, 1941 complaining of loss of hair during the past year. The family and past personal histories are irrelevant. The patient's general health has been good. The alopecia gradually started at the parting of the hair. It then advanced to the front and sides of the scalp. The patient combs her hair as often as six or seven times a day.

There are irregular, ill defined, diffuse areas of partial alopecia on the scalp, most conspicuous in the right parietal and frontal areas. In the affected portions

the hair is ¼ to ½ inch (0.6 to 1.3 cm.) long and appears to be broken off or cut short. There are no bare spots. The hair of the entire scalp is dry but is not easily pulled out. On examination with a magnifying glass the hairs seem to be of uniform diameter but show some undulation. There is no evidence of atrophy or inflammation of the scalp. The axillary and pubic hairs are normal. The integument, including the nails, appears normal except for keratosis pilaris on the thighs.

Routine laboratory tests, including mycologic examinations, gave negative results. Microscopic examination (Dr. Royal M. Montgomery) showed that the hair was curled slightly in places along its shaft. Near the distal ends of the undulated hairs there were definite twisting and some beading. The beading was not like that of monilethrix.

DISCUSSION

Dr. George M. Lewis: Besides the patchy alopecia, there were dark small dots scattered over the scalp. I think that another culture would show Trichophyton violaceum.

Dr. Herman Sharlit: I saw the evidence Dr. Lewis pointed out.

Dr. David Bloom: The patient admitted to me the habit of rubbing and scratching her scalp. I consider, therefore, that the condition is trichotillomania or trichokryptomania.

Dr. Fred Wise: The patient stated that she did not pull her hair. I was unable to detect any signs of ringworm infection.

A Case for Diagnosis (Tinea Due to Trichophyton Purpureum? Erysipeloid?). Presented by Dr. Fred Wise.

A. M., a woman aged 23, registered at the New York Skin and Cancer Hospital on Jan. 13, 1942 complaining of a pruritic eruption on the right hand of nine months' duration.

The initial lesion appeared last May under the nail of the right middle finger and on the subungual fold, which became red and itchy. The rash gradually spread along the middle finger to the dorsal surface of the hand and to the knuckles and finally extended to the second, third and fourth interdigital spaces. The lesions have been spreading slowly but continuously and have never regressed. It took about one month to involve a ½ inch (1.3 cm.) area. The patient states that an occasional pinhead-sized blister would form but would disappear spontaneously without bursting.

The nail of the right middle finger is deformed and is only one-third its original size. The remnant of the nail is fairly smooth, but it is thickened with a collection of debris under the free edge. On the dorsal surfaces of the middle finger and the knuckles and in the second, third and fourth interdigital spaces of the right hand are diffused superficial erythematous patches which end by a well demarcated line in the middle of the outer surface of the index and middle fingers. The same margins continue from the fingers in an unbroken manner and end in a festoon-like, distinctly raised border about ½ inch above the knuckles. Diascopic pressure obliterates entirely the reddened zone and reveals a uniform white color. The lesions are dry, not infiltrated and nontender. There is no atrophy and no telangiectasia. There is slight scaling between the toes. Microscopic examination of the scales of the hand revealed fungus.

A histologic section taken from the festooned border of the lesion on the hand was examined by Dr. Charles F. Sims. The description is as follows: "The epidermis is moderately and regularly acanthotic. The horny layer is thin and for the most part adherent to the surface of the epidermis except in the center of the section. In this location it is parakeratotic. The vessels of the upper part of the cutis are moderately dilated and surrounded by a sparse cellular infiltration composed of small round cells."

DISCUSSION

DR. GEORGE M. LEWIS: Clinically the eruption suggests invasion by Trichophyton purpureum, the color, configuration and lack of vesiculation being points in favor. There is also evidence of infected toe nails.

DR. MAX SCHEER: I think the eruption is tinea.

DR. MAURICE J. COSTELLO: The administration of sulfathiazole would be a good therapeutic test in differentiating this eruption from erysipeloid. It would cure the latter.

DR. E. WILLIAM ABRAMOWITZ: I favor the diagnosis of tinea.

DR. DAVID BLOOM: To me the lesion looks more like tinea than erysipeloid because of its color and the sharp, steep, raised border.

Pseudoxanthoma Elasticum with Angioid Streaks, Improved by Liver Therapy. Presented by DR. MAURICE J. COSTELLO.

A. D., a man aged 47, a Hungarian from Bellevue Hospital, presents an eruption of five years' duration. It involves the collar area of the neck, the supraclavicular areas, both axillas and the right cubital space. These areas consist of grayish linear and punctate reticulated, faintly yellow lesions, associated with loss of elasticity, as is evidenced by lack of recoil when the skin is stretched. The patient's chief complaint is rapid, progressive loss of vision. He also has mild pruritus. Angioid streaks can be detected in both retinas, especially the left. Histologic examination of skin from the affected area of the neck confirmed the diagnosis.

The Wassermann reaction of the blood was negative. He has received crude liver extract in 3 cc. doses three times a week for the past six months, with improvement in his general health, as is evidenced by increase in weight and strength. The pruritus of the involved areas has disappeared, and there has been definite improvement in visual acuity.

DAVID L. SATENSTEIN, M.D., *President*

ANTHONY C. CIPOLLARO, M.D., *Secretary*

Feb. 10, 1942

Radiodermatitis in a Case of Neurodermatitis. Presented by DR. DAVID BLOOM.

D. M., a woman aged 21, has been suffering from generalized neurodermatitis for nine years.

The dry, erythematous, lichenified and excoriated eruption is particularly pronounced in the cubital and popliteal fossae and on the face and scalp. The upper portion of the chest, the neck, the upper portion of the back and the cheeks present diffuse telangiectasia, atrophy and pigmented spots.

The patient had been treated by a general radiologist during 1939 and 1940. In May and June of 1941 she was treated by a dermatologist, who discontinued roentgen ray treatment after the sixth exposure. At this time radiodermatitic changes were noted on the chest, back and neck.

DISCUSSION

DR. E. WILLIAM ABRAMOWITZ: Cases of this kind are getting to be less prevalent. Patients who have received roentgen ray treatment may be incubating a radiodermatitis at the time they come for further treatment. Personally, I do not like to give further roentgen ray therapy to a patient who has been treated

by a general roentgenologist. I think it would be a good plan to limit roentgen ray therapy to not more than one or two courses in treatment of chronic recurrent eczematous eruptions. I should like to hear some suggestions as to treatment for the roentgen ray dermatitis. A vitamin A and D ointment may be useful. Thorium X paste has been used abroad. Plastic surgical treatment is not necessary.

Dr. Samuel Peck: Fractional doses of roentgen ray (75 r, unfiltered) given by a competent, well trained dermatologist have rarely, if ever, to my knowledge, led to undesirable sequelae. It seems to me that roentgen ray specialists tend to give much larger doses than a dermatologist would think necessary. I do not think that the use of roentgen rays in psoriasis can be condemned in all instances. The indications for this therapeutic procedure depend on the experience and judgment of the dermatologist. I should not presume to lay down any hard and fast rules for the use of roentgen rays in the treatment of psoriasis, except to say that it is inadvisable to use them for a widespread eruption or, certainly, before other methods of treatment have been given a thorough trial. In certain localizations, such as pararectal regions, the process may yield to other methods of treatment, even though the results are only temporary.

Dr. Maurice J. Costello: One occasionally sees radiodermatitis following roentgen ray treatment given by the dermatologist who has no knowledge of previous roentgen ray treatment. Generally radiologists who are not dermatologists unwittingly cause such conditions by administering excessive doses of roentgen rays. The patient is at fault most of the time, because he shops around and does not tell that he had received roentgen ray treatment previously. Many times this important information is withheld by the patient because he fears that he may be deprived of the rapid relief which often follows therapy with roentgen rays in chronic dermatoses.

Dr. Franklin Grauer (by invitation): It is tempting to treat patients with neurodermatitis with roentgen rays, especially when discomfort and a great deal of itching are present. Sometimes the patient will try almost to force one to use this type of therapy against one's better judgment. I think that whenever possible the use of topical remedies and sedatives should be relied on for such conditions and irradiation should be held in reserve. With regard to the treatment of radiodermatitis, in addition to previously accepted measures, I understand that good results have been obtained by administering large doses of vitamin B complex by mouth in conjunction with intramuscular injections of liver extract.

Dr. Howard Fox: I agree that in the large majority of cases of radiodermatitis, the roentgenologists and not the dermatologists are at fault. This is especially true of the past dozen years.

Dr. Max Scheer: Neurodermatitis in my experience is cured more rapidly with coal tar than with roentgen rays. Recurrences are not infrequent after either method. Neurodermatitis will be relieved by coal tar in an average of one week; with roentgen ray treatment the time is three to six weeks. I neither condone nor condemn giving ⅛ skin units (37 r, unfiltered) of roentgen rays once weekly for about a month. That depends on the dermatosis, the dryness or oiliness of the skin, etc. For all patients I try to get a history of previous roentgen ray treatment, including the number of exposures, the intervals, the time of the last exposure and the dosage (from the physician who administered the therapy). I am also guided by the condition of the skin. If the sebaceous secretion is normal or excessive roentgen ray treatment may be given if indicated. If the skin is dry (absence or diminution of sebaceous secretion) I omit roentgen ray treatment. In nearly all the cases of radiodermatitis I have encountered in private practice the condition has been caused by treatment administered by roentgenologists, in only a few was it caused by general practitioners (because they rarely use roentgen ray therapy). In recent years, i. e., the past ten or twenty years, I have not seen radiodermatitis produced by a dermatologist, though twenty-five or more years ago there were cases of this condition. I attribute this to the mathematical method of dosage first used and recommended by Dr. George M. MacKee.

DR. DAVID BLOOM: This case is presented in order to show again the risk of radiodermatitis in cases of those cutaneous diseases which are treated by the general radiologist. It should serve also as a lesson to the dermatologist not to expose any patient to roentgen rays before the exact amount of previous roentgen ray treatment has been ascertained.

Prurigo Nodularis. Presented by DR. E. WILLIAM ABRAMOWITZ.

B. F., a woman aged 45, a Russian Jewess, came to the New York Skin and Cancer Hospital on Jan. 14, 1942 with an eruption on the legs of five years' duration. She gave a history of having had high blood pressure, for which she has been receiving medication for the past three years.

At present the patient presents on the extensor surfaces of the legs and feet and to less extent on the flexor aspect of the legs pea-sized to hazelnut-sized, firm and somewhat crusted nodules, which are raised above the surface of the skin. These are associated with severe pruritis. One lesion is on the right sole.

Routine examination of the urine showed nothing abnormal. A blood count was normal except for showing mild leukocytosis. The histologic picture of a nodule "could fit in with prurigo nodularis."

DISCUSSION

DR. ISADORE ROSEN: The clinical features which are present in this patient do not coincide with those which are seen in prurigo nodularis. She has lesions which are distinctly inflammatory, and most of them have excoriated surfaces. In my experience, although itching is severe in prurigo nodularis, there are no excoriations.

DR. FRANKLIN GRAUER (by invitation): I think this case is one of prurigo nodularis. It might be interesting to stain sections from this tissue with the Masson trichrome stain to see whether a hyperplasia of nerve fibers can be demonstrated, which according to the findings of Pautrier is characteristic for this type of lesion.

DR. MAX SCHEER: I think the condition is prurigo nodularis.

DR. DAVID BLOOM: I favor the diagnosis of prurigo nodularis because the patient presents nodular firm lesions on the dorsa of the legs which have been present for many years and are associated with extreme pruritus.

DR. HOWARD FOX: I think this condition is undoubtedly prurigo nodularis. The sex and age of the patient and the location of the eruption are what one would expect in this disease. The lesions are discrete, extremely pruritic papules (or nodules), and their lack of tenderness would aid in excluding a diagnosis of bromoderma or iododerma.

DR. FRED WISE: According to Pautrier, the differentiation between prurigo nodularis and diseases which simulate it may readily be made by special staining of the nerve elements within the nodule of prurigo nodularis, the existence of which is said to be characteristic.

DR. E. WILLIAM ABRAMOWITZ: All the positive points in favor of prurigo nodularis have been mentioned, including the histologic picture. I want to bring out an additional point against bromoderma, and that is the fact that no new lesions have appeared in all these years.

Lupus Vulgaris; Tuberculous Adenitis. Presented by DR. GEORGE M. LEWIS.

Mrs. L. H., a Hungarian widow aged 72, had lesions on the lower part of the right cheek twenty years ago. Shortly after the rash was first noticed, a lump appeared on the right side of the neck. The eruption has slowly increased in extent. The lump remained about the same size until the last two weeks, during which time it has enlarged. The family physician suspected carcinoma and referred the patient to the Memorial Hospital for the Treatment of Cancer and Allied

Diseases, where a biopsy was performed on a specimen from the area of eruption and on an aspiration specimen from the tumor. The patient's second husband died from pulmonary tuberculosis.

On the lower part of the right cheek is an irregular, bright red eruption which measures approximately 5 by 3 cm. There is some central scarring. On diascopic pressure, definite apple jelly nodules may be seen. There is a large nonpainful lymph node in the right anterior cervical region. The overlying skin is unchanged and is not painful or tender.

The histologic diagnosis was lupus vulgaris. Biopsy of the aspiration specimen revealed tuberculosis. The Wassermann reaction of the blood was negative.

DISCUSSION

DR. MAX SCHEER: I think the condition is a typical lupus vulgaris.

DR. MAURICE J. COSTELLO: Judging from the cases of cutaneous tuberculosis I am observing at Bellevue Hospital, it is possible for tuberculous adenitis to develop at any age, although in the majority of cases it occurs early in life. If this patient does not have pulmonary tuberculosis now, it may eventually develop, because in many of the patients with long-standing lupus vulgaris well circumscribed lesions of rather inactive pulmonary tuberculosis develop with the appearance of tubercle bacilli in the sputum.

DR. GEORGE M. LEWIS: The adenitis appeared shortly after the beginning of the lupus vulgaris, that is, twenty years ago, and apparently it has increased during the past two or three months.

Herpes Zoster Following Administration of Mapharsen. Presented by DR. MAURICE J. COSTELLO.

E. B., a man aged 61, from Bellevue Hospital, was presented before the Section of Dermatology and Syphilis of the New York Academy of Medicine in November 1941 and in January 1942 (ARCH. DERMAT. & SYPH. **45**:1026 [May] 1942). After the third injection of 0.04 Gm. of mapharsen pain developed in the lumbar region on the right side, which was followed by the development of typical grouped herpetic vesicles following the course of the spinal nerves. The patient has latent syphilis and healed pulmonary tuberculosis.

DISCUSSION

DR. PAUL E. BECHET: The incidence of herpes zoster following the use of arsenical preparations in the treatment of syphilis is interesting, but reports of this incidence have not as yet been frequent enough to prove that this relation definitely exists; consequently a coincidence cannot be entirely eliminated. It is important, however, to report every case in order to arrive at some future definite conclusion.

DR. MIHRAN B. PAROUNAGIAN: I have observed a number of cases in which zoster followed treatment with arsenicals before mapharsen was introduced; so arsenic may be a factor in some cases.

DR. FRANKLIN GRAUER (by invitation): Arsenic, as is known, can produce herpes-zoster-like eruptions, and the condition in this case apparently is an example of this. I have always wondered whether in all cases of zoster the disease was not essentially on an infectious basis and whether in the symptomatic conditions the implicated drug or toxin did not act as a predisposing or "trigger" mechanism rather than as the exciting cause.

DR. E. WILLIAM ABRAMOWITZ: Hutchinson, Hebra and others believed in the causal relation between arsenic and herpes zoster. Whether this action is direct or due to the activation of a virus is still a debated question. I think arsenic as well as several other drugs may produce zoster.

DR. DAVID BLOOM: This case is presented for the purpose of showing that mapharsen like other arsenicals can cause zoster.

DR. HOWARD FOX: It has long been known that arsenic is a neurotropic drug, the administration of which may be followed by herpes zoster. This has happened too often to be a coincidence. It is reasonable to consider that this eruption was caused by mapharsen, a number of such eruptions having been observed at Bellevue Hospital and elsewhere.

DR. MAURICE J. COSTELLO: I think that whenever zoster is painless and not extensive one should suspect arsenic as the cause. When zoster is generalized or extensive with aberrant vesicles, Hodgkin's disease or one of the lymphoblastomas should be considered. Not infrequently patients with that disease have zoster. In my experience, zoster follows administration of the arsenicals in the following order of frequency: (1) solution of potassium arsenite U. S. P., (2) mapharsen, (3) silver arsphenamine and (4) neoarsphenamine. The point that impresses me is that the postarsenical herpes zoster is not as extensive and not as painful as herpes zoster of different causation.

Multiple Epithelioma Superimposed on Lupus Erythematosus. Presented by DR. MAURICE J. COSTELLO.

J. M., a policeman aged 35, has suffered from lupus erythematosus of the face and lower lip for the past nine years. This has been refractory to most types of therapy, including administration of gold sodium thiosulfate and of bismuth salicylate, local injections of 1 per cent gold sodium thiosulfate in 2 per cent procaine hydrochloride with epinephrine hydrochloride 1:15,000, refrigeration with solid carbon dioxide and cauterization with the electric cautery. In 1940 a prickle cell epithelioma was removed by surgical excision from the left side of the vermilion border of the lower lip. The diagnosis was confirmed by biopsy. A year later another similar lesion was removed from the right side of the lower lip.

He now presents a coffee-bean-sized infiltrated nodule on the inner aspect of the lower lip and a number of precancerous keratoses secondary to lupus erythematosus in this area.

DISCUSSION

DR. J. FRANK FRASER: It is important to recall the fact that lupus erythematosus of the skin or adjacent mucous membranes is not one of the diseases which is generally accepted as a precancerous dermatosis. The incidence of malignant transformation in lupus erythematosus is comparatively rare. As a matter of fact, it is no greater than would be expected when one considers the age of the patient and the action of various agents which are known to be carcinogenic. In my opinion the great majority of cases in which epithelioma has developed on lupus erythematosus have been those in which there was exposure to some carcinogenic agent, usually radium or roentgen rays. A patient with lupus erythematosus of the face and ears of twenty-seven years' duration was recently referred to me for diagnosis of an ulcerated lesion which had developed on a radium keratosis. The patient was treated with radium for lupus ten years ago at the clinic of the New York Hospital. When I saw him, examination showed chronic radiodermatitis with telangiectasis, atrophy and radium keratoses. My experience has been similar with lupus vulgaris. All the epitheliomas I have seen in this disease were preceded by roentgen ray therapy. Montgomery (Precancerous Dermatosis and Epithelioma in Situ, ARCH. DERMAT. & SYPH. **39**:387-408 [March] 1939) stated: "Veiel found the occurrence of epithelioma to be less frequent in lupus erythematosus than in lupus vulgaris." In Dr. Costello's case it is apparent that other factors than the roentgen ray treatment must be considered.

DR. HOWARD FOX: I have always understood that epithelioma developed in about 1 to 2 per cent of the cases of lupus vulgaris, but my impression has been that the incidence of the development of cancer was much lower in lupus erythematosus.

DR. MAX SCHEER: I agree with Dr. Fox that cancer occurring in lupus erythematosus is rare. In lupus vulgaris it is not at all uncommon and especially so if the patient has been treated with roentgen rays. This patient has been a

heavy smoker (mostly of cigarets), and it is possible that his excessive indulgence in tobacco has a bearing on his epithelioma.

DR. E. WILLIAM ABRAMOWITZ: Some patients with lupus have cancer and some do not. I do not believe any definite cause has yet been proved. I have observed several cases of lupus erythematosus in which an affected area was epitheliomatous. In 1 instance extensive destruction of the nose followed. This patient has signs of lupus erythematosus around the lesion, and therefore I do not think that he should have roentgen rays or radium. I think it would be best to perform a plastic operation, as the tissues are not as vital as they should be.

DR. MIHRAN B. PAROUNAGIAN: I have seen epithelioma develop in patients with lupus erythematosus who were treated with solid carbon dioxide and other irritants.

DR. MAURICE J. COSTELLO: When this patient was first examined at another hospital the correct diagnosis was not made immediately, and he received several roentgen ray treatments. As this occurred over five years ago, I do not think that this small amount of superficial fractional treatment had any bearing on the subsequent development of the prickle cell epitheliomas. Since he was a patrolman and was exposed to the elements, I tried to arrest the progress of this disease somewhat by having him work indoors. Keratoses frequently develop on the lower lip, and if they are not removed immediately an epithelioma develops. He had three such lesions in the past three years. I think that this area bearing precancerous lesions should be completely excised.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILIS

LOUIS TULIPAN, M.D., *Chairman*

TIMOTHY J. RIORDAN, M.D., *Secretary*

Jan. 6, 1942

A Case for Diagnosis (Dermatitis Medicamentosa? Papulonecrotic Tuberculid? Leukemid?). Presented by DR. LEWIS B. ROBINSON.

F. M., a woman aged 56, is presented from Vanderbilt Clinic with an eruption on the backs of the hands and fingers and on the left elbow of one year's duration. After an attack of poison ivy dermatitis in August 1940, occasional small red papules began to develop on the hands and on the left arm, which lasted for several months. She was admitted to the clinic in January 1941, at which time she had been taking diurbital (a proprietary preparation, each tablet of which contains 3 grains [0.18 Gm.] theobromine with sodium salicylate, ¼ grain [0.015 Gm.] phenobarbital and 1½ grains [0.09 Gm.] calcium lactate) and theophylline with ethylenediamine for six months. The administration of these drugs was discontinued, with no change in the eruption. She has since taken an occasional dose of theophylline with ethylenediamine.

The lesions have become more numerous and seem to appear in crops. They are small red firm papules which are sometimes tender. They seldom completely disappear, although there are a few scars.

Sections of a small piece of skin from the left arm disclosed an epidermis which was not increased in width. The rete pegs were absent. Hyperkeratosis was present but not extreme. There was some degenerative change in the corium, with a striking lymphocytic infiltration about the blood vessels. This extended into the subcutaneous fat. There was no distinct evidence of erythema induratum. It was felt that lupus erythematosus of the deep type, dermatomyositis and one of the lymphoblastic infiltrations should be considered. A blood count and exam-

ination of the urine for sugar were recommended, with panniculitis to be considered as a further possibility.

A blood count in January 1941 showed red cells 4,400,000 and white cells 4,200, with polymorphonuclear leukocytes 38 per cent, small lymphocytes 53 per cent, large lymphocytes 3 per cent and eosinophils 6 per cent. The hemoglobin content was 80 per cent. In May 1941 the differential count revealed 4,500 white blood cells, with polymorphonuclear leukocytes 27 per cent, small lymphocytes 65 per cent, large lymphocytes 3 per cent and eosinophils 5 per cent. The hematologic reports indicate a persistent mild secondary anemia, but there has been nothing suggestive of leukemia. The basal metabolic rate was — 19 per cent.

DISCUSSION

DR. E. WILLIAM ABRAMOWITZ: This patient is having her hair dyed, probably with a paraphenylene dye or one related to it. Such compounds are known to be toxic. They cause a contact dermatitis and in some instances a purpura. It is possible that the present eruption is due to the hair dye.

DR. J. GARDNER HOPKINS: Perhaps one makes the diagnosis of papulonecrotic tuberculid somewhat glibly, but one often feels fairly sure of this diagnosis on circumstantial evidence when the biopsy shows no tuberculous structure. In this case the lesions above the elbow do not seem typical of papulonecrotic tuberculid, but I can think of no more probable diagnosis. I should like to ask Dr. Abramowitz whether little papular lesions with necrosis in the center are seen in drug eruptions and also whether drug eruptions from theophylline with ethylenediamine have been encountered.

DR. E. WILLIAM ABRAMOWITZ: Nodular eruptions from drugs like antipyrine, the barbiturates, the bromides, the iodides, the salicylates and sulfathiazole are not unusual. No necrosis is present clinically or histologically in such lesions. Regarding eruptions due to theophylline with ethylenediamine, the late Dr. Feit presented a patient with an erythema-multiforme-like eruption from theominal, a proprietary preparation of theobromine and phenobarbital (ARCH. DERMAT. & SYPH. **34**:320 [Aug.] 1936). Theophylline with ethylenediamine is pharmacologically and chemically related to theobromine, and it is possible that it may cause similar eruptions and also nodose lesions.

DR. FRED WISE: I can only express the general impression that this eruption would conform with that of papulonecrotic tuberculid. It is my understanding that the patient has had the eruption for three years, that she has stopped taking any drugs and that the condition has grown worse. I should perform guinea pig inoculations and biopsies in order to obtain more information.

DR. MAURICE J. COSTELLO: Have graded intradermal tests with tuberculin been performed? If I had one choice, I should make a diagnosis of papulonecrotic tuberculid. I do not think it is unusual to find nodose or erythema-induratum-like lesions in a patient with papulonecrotic tuberculid. They usually occur around the ankles and feet.

DR. LOUIS TULIPAN: I noticed that this patient had a few scars on the posterior part of the legs, which is the usual location for the lesions of erythema induratum. I believe the lesions seen in other parts would fit the clinical picture of papulonecrotic tuberculid, and I think the disease is some kind of tuberculid, in spite of the histologic report. I believe other lesions should be studied.

A Case for Diagnosis (Erythema Annulare Centrifugum?). Presented by DR. LEWIS B. ROBINSON.

E. B., a man aged 57, is presented from the Vanderbilt Clinic with a generalized eruption. This began in 1938 with small itching areas on the backs of the hands which lasted all winter. In June 1938 the lesion spread in six weeks to involve the entire body. He was admitted to Long Island College Hospital, Brooklyn, for two weeks with diffuse swelling of the face and extremities and a

crusting and vesicular dermatitis. There were also symmetric, well circumscribed, pinkish areas with clear centers. The acute phase subsided, and he was followed in the clinic until April 1941, during which time he had repeated mild attacks of symmetric, circular lesions, mostly fading in the center. The diagnosis was erythema multiforme.

Tests for allergy with house dust, tomato and spinach elicited 2 plus reactions.

The histologic picture was compatible with syphilis but not with sarcoid or mycosis fungoides.

Treatment consisted of injections of anterior pituitary extract from October to December 1939, ultraviolet irradiation from January to May 1940 and Staphylococcus aureus and Bacillus coli vaccine from January to April 1941.

An acute exacerbation occurred in May 1941, and the patient was admitted to Vanderbilt Clinic. The body showed multiple large and small circumscribed lesions of symmetric distribution, with raised pinkish borders and fading in the center and with a superimposed dermatitis from scratching and local applications.

The patient was given roentgen ray therapy, which relieved the acute phase, together with local therapy and intramuscular injections of liver extract. In June 1941 all the teeth showed root abscesses and were extracted. Since then the patient's condition has improved. New lesions have appeared on the neck, forearms and back.

Roentgen ray examination of the chest showed no abnormalities, and a gastrointestinal roentgen ray series was normal except for showing hypermotility of the stomach and intestines. A streptococcus sensitization test produced decided erythema, while the results were negative to staphylococcus. The differential blood count was normal.

DISCUSSION

DR. E. WILLIAM ABRAMOWITZ: This is an annular papular eruption of the erythema multiforme type. The patient takes a cough medicine which he inherited from his mother, and it would be worth while to find out what it contains.

Hepatosplenomegaly, Probably Due to Lymphogranuloma Venereum. Presented by DR. WILLIAM CURTH.

A. M., a man aged 45, is presented from Vanderbilt Clinic, for Dr. Helen C. Curth. He has been in the United States for the past eighteen years, and, with the exception of having the grip during the first world war, he has never been sick. Early in May 1941 pain developed in the left inguinal region, with severe headache. Two weeks later he consulted a private physician, who gave him capsules and an injection in the hip. He was admitted to Presbyterian Hospital on May 24, with a temperature of 103 F., a purpuric rash on both legs and an enlarged, firm, tender, nonfluctuant node in the left inguinal area. The axillary nodes were palpable. The liver was enlarged 3 fingerbreadths and the spleen 2 fingerbreadths. The genitals and the rectum were normal. The following day the reaction to a Frei test was positive, to a Ducrey test negative and to a Wassermann test of the blood anticomplementary. Agglutination tests on a blood specimen were negative for typhoid, paratyphoid A and B, typhus and tularemia organisms and for Brucella. A smear for malarial organisms was negative. The complement fixation test for lymphogranuloma venereum in a dilution of 1:240 gave a strongly positive reaction.

The blood count on May 24 showed 4,600,000 red cells and 10,400 white cells, with polymorphonuclear leukocytes 67 per cent, lymphocytes 23 per cent, monocytes 8 per cent and eosinophils 2 per cent.

There was no jaundice and no ascites. Sulfadiazine (2-[paraaminobenzenesulfonamido]-pyridine) was given, with no improvement. The patient was then given sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) for about ten days. The temperature remained as high as 101 and 102 F. On June 2 the bubo was aspirated, and this was repeated five times. The pus was used each time as an antigen, which elicited positive reactions in known cases of lymphogranuloma

venereum and negative reactions in control cases. After being given intravenous injections of Frei antigen and repeated aspirations, the patient first had a low grade fever and then became afebrile. He was discharged on July 11, at which time the liver and spleen remained large. He stated that he was not addicted to the use of alcohol.

Examination at present still shows the liver to be enlarged 9 cm., smooth, hard and nontender and the spleen to be enlarged 2 cm. In the left inguinal area fibrous nontender tissue can be felt, with the skin unchanged. The right inguinal area is normal.

The reactions to Frei tests both with human antigen and with chick embryo antigen (lygranum) are still strongly positive. The Wassermann reaction was negative on June 12. The results of various tests from May 1941 to the present are given in the accompanying table.

Results of Tests in Case of Hepatosplenomegaly

Date	Protein, Gm. per 100 Cc. Serum			Cephalin Flocculation	Bromsulphalein, per Cent
	Total	Albumin	Globulin		
5/26/41	.....	.....	.....	4 plus	....
5/27/41	7.8	2.9	4.9	.....	....
5/29/41	.....	.....	.....	.....	35
6/ 3/41	9.0	2.7	6.3	4 plus	....
6/10/41	.....	.....	.....	4 plus	....
6/24/41	8.8	3.2	5.6	3 plus	....
6/26/41	.....	.....	.....	.....	15
10/ 7/41	7.6	4.4	3.2	1 plus	15
1/ 2/42	.....	.....	.....	negative	10

DISCUSSION

DR. F. REISS (by invitation), Shanghai, China: I have had opportunity to observe cases of lymphogranuloma venereum and on extremely rare occasions have seen hepatomegaly and splenomegaly, but never to such an extent as in this case. I should like to ask whether an examination of the fundi has been made and whether the albumin-globulin ratio was inverted.

DR. J. GARDNER HOPKINS: A larger variety of lesions are gradually coming to be recognized as being due to the virus of lymphogranuloma venereum. However, as in all nonfatal diseases, one is confronted with the impossibility of proving the cause of visceral lesions in the individual case. I do not see how one can be certain in this instance, but it seems important to record the diagnosis as lymphogranuloma venereum accompanied by enlargement of the liver and spleen. I think it should be assumed that hepatomegaly and splenomegaly can be caused by this virus.

DR. CHARLES WOLF: This patient comes from a region where echinococcus cysts are prevalent. It might be advisable to consider that as a possibility.

DR. E. WILLIAM ABRAMOWITZ: Has the enlargement of the liver and spleen receded under treatment?

DR. HELEN O. CURTH (by invitation): The size of the liver has not decreased, but the functional changes have disappeared. It is hard to know whether the changes are due to the infection with lymphogranuloma venereum, especially because the condition of the liver and spleen in the patient prior to the beginning of the disease is not known. In Kornblith's case of hepatosplenomegaly (*J. Mt. Sinai Hosp.* **3**:24 [May-June] 1936) the liver and spleen returned to normal size when the inguinal adenopathy cleared up. In the patient described by Chapman and Hayden (*New England J. Med.* **217**:45, 1937) the spleen was enlarged. All patients with this condition suffered from purulent inguinal adenitis, with

severe systemic reactions. Our patient was acutely sick. A diagnosis of septicemia was made at first.

With regard to the differential diagnosis, it should be noted that an inverted albumin-globulin ratio is also seen in cirrhosis of the liver. The rapidity, however, with which the changes in this case took place has never been observed in a case of cirrhosis of the liver. The globulin increased in six days from 4.9 to 6.3 Gm. per hundred cubic centimeters. The gradual improvement of the liver function would also point against cirrhosis of the liver, which is a progressive disease, although fluctuations do occur in that condition. Cutaneous and complement fixation tests for echinococcus should have been made and were considered. However, the absence of eosinophilia would speak against that diagnosis.

The fact remains that the man had acute lymphadenitis due to lymphogranuloma venereum. The inguinal adenitis and the disturbance of the liver function subsided; at least, the chemical evidence would bear that out.

I should like to make a plea, especially to the dermatologists, because they are the ones who observe the early stages of the disease, for studies of the liver and spleen in patients with lymphogranuloma venereum.

Trichorrhexis Nodosa. Presented by DR. A. BENSON CANNON.

R. O., a girl aged 18, is presented from Vanderbilt Clinic for Dr. Jerome K. Fisher. She has been well all her life but felt she was overweight; two years ago, therefore, at the age of 16, she went on a restricted diet. This consisted of coffee for breakfast, no lunch and lettuce and tomatoes for supper, with an occasional orange or glass of milk. She stayed on this diet for one year without losing weight. The menstrual periods, which had been regular since the age of 13, now stopped and have been irregular since then. There has been no recent change in weight.

The patient first came to the clinic in October 1941. Two years previously she had noticed thinning of the hair, and fourteen months ago she noted two bald spots on the vertex about the size of a quarter. The scalp was not bald on these areas, but the hair was short. The spots gradually enlarged until they covered the entire vertex.

The patient says she does not pick at the hair or wear any sort of hat except a kerchief at times. She has had no beauty parlor treatments. She shampoos her head about twice a month and has used a patent hair tonic. When the baldness appeared she consulted physicians, four in all, who have given her oils for the hair, injections in the scapular regions "to make the hair grow" and injections in the buttocks, which brought on her menses. She has also taken thyroid. The patient is easily upset and bites her nails. The menses are still irregular.

Examination reveals the hair to be dry and rough. On the vertex of the scalp over an area about 12 to 15 cm. in diameter the hair is 1 to 2 cm. long and is stiff, dry and rough. The scalp is not involved. The remaining hair is about 15 to 20 cm. long.

Microscopic examination shows hairs from the axillas, pubis and arms to be normal, while hairs from the thinning area of the vertex and also the long hairs on the head show nodular, brushlike fractures and splintering.

A biopsy of tissue from the scalp revealed no pathologic change. The basal metabolic rate was —9 per cent. The Wassermann reaction of the blood was negative.

Since Oct. 29, 1941 the patient has received from 75,000 to 100,000 U. S. P. units of vitamin A daily by mouth. The hair has grown 1 to 2 cm., has regained its luster and has become softer.

DISCUSSION

DR. J. GARDNER HOPKINS: This brings up an interesting point in the causation of trichorrhexis nodosa. I have always thought of the disease as being the result of trichorrhexomania, and I believe it has been produced artificially in nor-

mal scalps by repeated rubbing. However, if Dr. Fisher can show that he can cure the condition with vitamin A, that idea will have to be abandoned.

DR. E. WILLIAM ABRAMOWITZ: I think that it was Sabouraud who said trichorrhexis nodosa was somewhat like a localized neurodermatitis of the scalp appearing in persons with a neurosis. If that is the case, one would think that vitamin B rather than vitamin A should be given.

DR. PAUL GROSS: The hair clinically and microscopically suggests trichorrhexis, but I have never seen as extensive and patchy an alopecia in a case of this disease.

DR. LOUIS TULIPAN: I recall having read in the Russian literature a report in which trichorrhexis nodosa occurred in some horses and also in the persons handling the horses. I have not read of similar cases occurring anywhere else, but this brings up a possible infectious cause for this disease.

Acrosclerosis (Multiple Telangiectases of the Face and Mutilation of Digits). Presented by DR. EUGENE TRAUGOTT BERNSTEIN and DR. LEWIS A. GOLDBERGER.

A. S., a woman aged 45, was first seen at Mount Sinai Hospital in December 1935 with a complaint of blue and infected fingers. She presented then what was thought to be a typical picture of scleroderma. A parathyroidectomy was performed in January 1936, but there was no improvement during the next twelve months. In January 1937 a right cervicothoracic ganglionectomy was performed, but this procedure was likewise not efficacious. Since 1938 she has been treated under the supervision of therapeutists at the peripheral vascular clinic. She received iontophoresis with mecholyl chloride (acetylbetamethylcholine hydrochloride) and then with histamine, with some improvement. She also received pancreatic tissue extract, with no results. The patient was readmitted to the hospital in December 1939, with a picture of scleroderma. She was given 1 cc. daily of a 0.5 per cent solution of dihydrotachysterol in oil; after one week of treatment improvement was noted, and this progressed with continued treatment. After four weeks of therapy the dose was reduced to 0.5 cc. daily.

Examination at present shows the face to have a masklike appearance; the skin is taut and rigid in part, but a certain soft quality has remained. There are multiple spider-like telangiectases on the nose and cheeks. There is shrinkage of the tissue of the hands and mutilation of the finger tips. The patient is unable to flex or extend the fingers completely.

Before treatment with dihydrotachysterol the face was tight, the mouth drawn and wrinkled and the lips thin. The patient could hardly open her mouth. After approximately two years of treatment the skin is softer and the atrophy much reduced. Mobility of the fingers is increased; the ulcerations are practically healed, and the patient feels greatly improved.

The calcium content of the blood serum varied from 9 to 13 mg. per hundred cubic centimeters during the time of administration of dihydrotachysterol.

Examination of the heart revealed a high-pitched systolic murmur, rasping at the apex and louder at the left interspace. The electrocardiogram showed deviation of the axis to the left. The patient suffers from precordial pain on exertion.

This case was previously reported by Drs. M. Perez, L. J. Soffer and S. Silbert, from the peripheral vascular clinic and the medical service of Dr. George Baehr (Improvement in Two Cases of Diffuse Scleroderma by Use of Dihydrotachysterol [A. T. 10], *J. Mt. Sinai Hosp.* **6**:333 [March-April] 1940).

DISCUSSION

DR. FRED WISE: How does the condition differ from Raynaud's disease?

DR. PAUL GROSS: I should like to know the rationale of treating patients with this condition with dihydrotachysterol.

DR. ANTHONY C. CIPOLLARO: I recently discussed diseases such as morphea, scleroderma, sclerodactylia and acrosclerosis with an internist. It was his opinion that these diseases are really vascular diseases and that similar disturbances might be found in other organs. He stated that the prognosis was generally unfavorable. This view is contrary to that held by most dermatologists except in cases of generalized scleroderma. It remains for future research to reveal whether or not a disease of this group causes death within a short time from inception.

DR. HERMAN GOODMAN: It would be interesting to observe the roentgenograms of the terminal phalanges of patients with conditions in the categories mentioned as including acrosclerosis. In the past it has been impossible to distinguish the osseous changes of the individual diseases named. Osseous changes of the terminal phalanges also occur in adrenal diseases and certain of the blood dyscrasias.

DR. J. GARDNER HOPKINS: The remark quoted by Dr. Cipollaro is typical of what happens when an enthusiast becomes interested in a disease with which he is not familiar. I do not think one need fear the prospect of early death from this condition. There are many patients with morphea and with acrosclerosis who have recovered completely.

DR. EUGENE TRAUGOTT BERNSTEIN: The purpose of presenting this case is to find out whether or not it is justifiable to consider acrosclerosis as a separate entity. The condition was first described by Sellei, who applied the term acrosclerosis to a syndrome which was formerly considered the sclerodactylia type of scleroderma. I think the disease should be accepted as a separate clinical entity on the basis of the mode of onset and the course.

In the case presented, the process arose on the hands and face symmetrically and conjointly and was associated with vasomotor changes of the Raynaud type. The process in acrosclerosis, as in the presented case, is one of shrinkage and contraction, which is discernible in the hands and face, while scleroderma is an infiltrative process. The changes progressed in this patient, and the distal phalanges became shorter and sharper until mutilation developed. The skin of the face also shrank to such an extent that it produced a masklike appearance, but in spite of this a certain soft quality of the skin remained, with telangiectases. I am therefore of the opinion that this patient has vascular changes resulting in pronounced spider-like telangiectases, a heart murmur and Raynaud's syndrome.

The exact mechanism of the therapeutic effect of dihydrotachysterol is unknown in cases of scleroderma. Its use is purely empiric.

CHICAGO DERMATOLOGICAL SOCIETY

LEONARD F. WEBER, M.D., *President*

MICHAEL H. EBERT, M.D., *Secretary*

Jan. 21, 1942

A Case for Diagnosis (Dermatitis Herpetiformis?). Presented by DR. JAMES H. MITCHELL and DR. FRANCIS W. HETREED (by invitation).

E. K., a woman aged 64, stated that six years ago small, hard, itching growths appeared on her arms and on one small area on the back. Two years ago the eruption began to spread over the neck, chest, abdomen and back. The itching has remained constant and severe.

When the patient was first seen, three years ago, she had pea-sized verrucous nodules on the extensor surfaces of the arms and legs. The Kahn reaction, results of chemical examination of the blood and the differential blood cell count were normal at that time.

She now presents excoriated, slightly bluish papules and nodules on both extremities. Some of the lesions on the hands have undergone involution, leaving a hypertrophied scar. On the sternum and in the scapular and sacral areas as well as on the abdomen are grouped, roughly symmetric areas of excoriated papules. Scars are also present.

Treatment in the past has consisted of the administration of acetarsone and sulfanilamide, besides various topical applications.

At present the Kahn and Wassermann reactions are negative. Examination of the blood revealed 58 per cent hemoglobin, 3,180,000 erythrocytes and 9,450 leukocytes, with a differential count of 77 per cent neutrophils, 21 per cent small lymphocytes and 2 per cent eosinophils. Chemical examination of the blood showed nonprotein nitrogen 49 mg. and creatinine 1.6 mg. per hundred cubic centimeters.

DISCUSSION

Dr. Richard S. Weiss, St. Louis: This eruption is an extremely severe dermatitis herpetiformis. There are a few lesions that seem to be granulomatous and suggest papulonecrotic tuberculid, but I am sure that that diagnosis can be excluded.

Dr. H. E. Michelson, Minneapolis: I do not know what type of tuberculid Dr. Weiss is referring to, but I am positive that I have seen neither a written description nor a clinical example of a tuberculid that looks like the eruption in the presented case.

Dr. Louis A. Brunsting, Rochester, Minn.: I believe the patient has dermatitis herpetiformis. Recently in a general discussion of the treatment of this disease Dr. Senear brought out the beneficial effects of sulfapyridine (2-[para-aminobenzenesulfonamido]-pyridine), and I have been able to confirm this in several instances. Sulfapyridine is used empirically and seems to be the only one of the sulfanilamide group of drugs to have this effect. In 1 striking case, that of a patient who has been under my observation for the past four months, the signs and symptoms of extensive dermatitis herpetiformis of twenty years' duration were cleared within a week by means of sulfapyridine given by mouth, but when administration of the drug was discontinued the eruption recurred. A maintenance dose of 1 to 2 Gm. daily controls the symptoms. The patient is a young man in his thirties with no other disabilities, and he seems to tolerate the drug well, but because of the known tendency of untoward reactions to occur he is being kept under close observation. He was given a trial treatment with nicotinic acid because it too contains the pyridine nucleus, but there was no benefit from this treatment.

Dr. Francis E. Senear: My discussion that Dr. Brunsting referred to was based on experience with but 1 case, that of a little girl who was brought here from another city. She had had sulfanilamide previously without benefit but responded to sulfapyridine, the latter being the only other drug in that group available at that time. The child's lesions recurred promptly with cessation of the drug and cleared when administration of the drug was resumed. I have used it in several cases since then, with the same results as indicated by Dr. Brunsting: It helps in some cases and does not in others. I have not given any patients long-continued maintenance doses such as he mentioned.

Dr. Harry R. Foerster, Milwaukee: I agree with the presenter's diagnosis. One thing about the eruption that I think is of interest is the hyperkeratotic, large, flat papules, not unlike those seen in prurigo nodularis, which this patient has on the extensor surfaces of the extremities. I should like to know Dr. Mitchell's interpretation of those lesions. Could they be due to arsenical medication?

Dr. Theodore Cornbleet: In regard to the use of sulfapyridine, a man in Dr. Senear's service who has had dermatitis herpetiformis for many years gets excellent though temporary results with the use of sulfapyridine. One tablet for two days is sufficient to control the lesions and itching. If the sulfapyridine is not given for several days he again has an eruption and itching.

DR. S. W. BECKER: Some of the lesions which this woman presents resemble prurigo nodularis. I recently had a woman with severe diabetes and rather severe neurotic excoriations who presented lesions of this prurigo type. The microscopic section was suggestive, though not typical, of prurigo nodularis. In her case the lesions did not persist; some would heal, which does not occur in prurigo nodularis. Some persons react to chronic irritation in the form of prurigo-like lesions.

DR. CLARK W. FINNERUD: I understand that when this patient was first seen the lesions were different from those present today. I should like to know what they looked like at that time. I think it will be agreed that the eruption as it appears today is definitely dermatitis herpetiformis.

DR. JAMES H. MITCHELL: I saw this patient only once, last week. In the discussion the question of prurigo nodularis was mentioned in the differential diagnosis. It was because of the resemblance of the patient's condition to that disease that we thought it might be of interest to show the patient. Biopsy was performed three years ago, and Dr. Hetreed has had the patient under observation from time to time since. In the clinic I expressed the opinion that at present I thought the diagnosis was dermatitis herpetiformis.

I should like to say one word with regard to the treatment of this disease. I had several patients who resisted all types of treatment. As vitamin B complex is almost as popular as sulfanilamide and its derivatives, I sought to give vitamin B complex and a bismuth compound. One patient whose disease had resisted every kind of therapy and who was in the worst possible condition has continued to remain well for the past year and a half with this therapy. I have tried having him stop taking one or the other drug, and he invariably has an exacerbation. Several other patients have done well with the same combination. I do not know whether this therapy has any merit or whether the improvement obtained is merely a coincidence.

DR. F. W. HETREED: When I first saw this patient, three years ago, she had pruriginous nodules, some of them verrucous, on the lateral aspects of the arms and legs. Hypertrophic lichen planus and prurigo nodularis were considered. The biopsy did not show the characteristic infiltrate of lichen planus. Today she has a typical dermatitis herpetiformis. We thought it would be interesting to present this case, as the cause of both conditions is still obscure; there is a possibility that this patient has two distinct clinical entities: dermatitis herpetiformis as well as prurigo nodularis.

A Case for Diagnosis (Lupus Erythematosus?). Presented by DR. I. M. FELSHER (by invitation) and DR. EDWARD A. OLIVER.

J. D., a married white woman aged 48, a patient at the clinic of the Northwestern University, presents an eruption consisting of large annular lesions on the right side of her face, in the submaxillary area and on the neck and two smaller coin-sized annular lesions, on the left side of her face and forehead respectively. The disease began as a small red papule on the right side of her face in October 1941.

When the patient was first observed in November 1941, she presented an erythematous, edematous, annular lesion, about 4 cm. in diameter, having a sharply defined, elevated border of a more vivid red, covered in part with thin scales and small vesicles. She also complained of severe itching and burning. Since then the lesion has spread peripherally, clearing to a large extent in the center but maintaining an annular border that extends at present over the areas mentioned. The smaller lesions, also red and annular with a definite border, appeared later and spread slowly peripherally. The patient states that at times the lesions flare up, becoming more swollen and red, with increased itching or burning. She noticed that exertion, excitement, fatigue and exposure to heat provoked an exacerbation.

In 1936 she had a hysterectomy and a thyroidectomy, and since then her weight has been reduced 50 pounds (23 Kg.).

Examination for fungi and Kahn tests of the blood gave repeatedly negative results. The findings on urinalysis and the blood count were within normal limits.

The basal metabolic rate since the thyroidectomy has varied from —30 per cent in 1937 to +14 per cent in November 1941. Potassium iodide produced no effect on the lesions.

The patient received injections of estrone (theelin) and oral doses of thyroid and phenobarbital at different periods in 1937 and 1938. During these years she frequently suffered from an erythematous eruption on the trunk provoked by excitement, exertion or heat. She was free from any dermatosis from 1939 until October 1941.

DISCUSSION

DR. L. M. WIEDER, Milwaukee: I am unable to make a diagnosis, but it seems to me that there are a number of features in favor of lupus erythematosus, namely, the location, the gradual extension of the lesions and the presence of some atrophy or pseudoatrophy on the right cheek. I could not get a history of use of any drug that seemed to have any definite relation to this condition. There are no features characteristic of self-inflicted lesions. I favor the diagnosis of lupus erythematosus.

DR. JOHN F. MADDEN, St. Paul: I think the eruption is either lupus erythematosus or annular sarcoid. I do not think a diagnosis can be made without a biopsy.

DR. S. ROTHMAN (by invitation): In 1916, under the heading of erythema annulare centrifugum, Darier described a toxic eruption consisting of rapidly spreading erythematous-edematous annular lesions, similar to those of the patient presented here. In 1 case I observed continuous spreading for several months without being able to find any toxic agent or allergen. Once the lesions disappeared suddenly and spontaneously but recurred later in other regions.

DR. MAURICE OPPENHEIM (by invitation): Unna described such a condition. He considered it to be superficial lupus erythematosus and gave it the name ulerythema centrifugum. I believe the condition in this case belongs to the group of diseases which Unna described under this name.

DR. WILLIAM A. ROSENBERG (by invitation): I questioned this woman as to whether she took any drugs. I wondered whether this condition and the exacerbations are not the result of the phenobarbital that she is taking.

DR. L. M. WIEDER, Milwaukee: I questioned this patient in detail concerning ingestion of drugs. She stated that she has not taken any laxative containing phenolphthalein for the past six months. The only laxative she has taken is magnesium sulfate.

Hemorrhagic Diathesis and Lichen Planus. Presented by DR. S. W. BECKER and DR. S. ROTHMAN (by invitation).

R. R., an American housewife aged 32, has been known to be a "bleeder" since early childhood. She was admitted to Billings Hospital on Feb. 15, 1940 with anemia, dehydration, extended suffusions and strong petechial response to the Rumpel-Leede test. Studies of the blood revealed a normal platelet count and normal clotting time but considerably prolonged bleeding time (thirty minutes). After having received six blood transfusions the patient left the hospital in good condition on April 12, 1940. The disease was diagnosed as hemorrhagic diathesis or nonthrombopenic purpura of unknown cause. Together with 3 similar cases her case has been discussed in a publication of G. Carpenter and J. G. Allen (*Am. J. M. Sc.* **202**:655, 1941).

Five to six weeks ago a few red itching spots appeared on both legs and on the left thigh. Itching has been particularly intensive at night.

Besides extended suffusions, there are round, brownish red lesions, 0.4 to 2.0 cm. in diameter, consisting of confluent, tiny hemorrhagic papules, some of them displaying a lichenoid shine. On top of the larger papules a hyperkeratotic thick scale can be seen. The smaller lesions have a whitish shiny surface similar to Wickham's striae.

Histologic examination of a section removed showed the characteristics of lichen planus.

DISCUSSION

DR. HARRY R. FOERSTER, Milwaukee: The lesions on the thighs are characteristic of lichen planus. Those on the legs are atypical because of the hemorrhagic features, which are no doubt due to the underlying blood dyscrasia.

DR. RUBEN NOMLAND, Iowa City: What was the nature of the pigment?

DR. S. W. BECKER: We have not performed a biopsy on the pigmented area in this case, but we have in other cases, in which similar dark gray plaques showed hemosiderin on section.

DR. CLARK W. FINNERUD: Although I did not see the section, I took it that the hemorrhagic element, though not ordinarily seen in lichen planus, might be accounted for on the grounds of stasis. I do not know whether the condition is lichen planus.

DR. HAMILTON MONTGOMERY, Rochester, Minn.: The sections show the typical picture of lichen planus. I did not see any deposits of hemosiderin, although this is hard to distinguish from melanin pigment without special stains. In ordinary types of lichen planus the pigment is melanin. Many of the lesions clinically are brown rather than bluish. However, there is a mixture of blue and brown in the lesions on the leg, and this can be explained on the basis that in practically all dyscrasias of the blood one may expect to find varying amounts of hemosiderin deposited in the skin.

DR. S. ROTHMAN (by invitation): There are punctate hemorrhages and typical lichen planus lesions on the buccal mucous membrane.

A Case for Diagnosis (Mycosis Fungoides?). Presented by DR. FRANCIS E. SENEAR.

About the middle of 1940 the patient, Mrs. C. E., noticed some erythematous lesions on the labia majora. Several months later several nodules had developed at the site of these lesions. In June 1941 one of these nodules was removed, and in July three small nodules were excised. The only information which the patient had with regard to the pathologic diagnosis was that no cancer was present. Soon after that a large lesion developed involving the perineum and buttocks, which she described as a "port wine" mark. Gradually an extensive infiltration developed involving the genitalia, perineum and buttocks. Ulceration eventually occurred in the infiltrate involving the lower portion of the vulva and the tissue immediately posterior, and this has persisted until the present time. In July 1941 a large erythematous lesion developed in the left temporal region, and similar lesions soon developed on the forehead and in the left retroauricular region. Within the past few months numerous lesions have appeared on the body. There has been no itching.

The eruption on the trunk shows a number of lesions varying in size from about 2 to 10 cm. in diameter; some of these are macules; others are infiltrated plaques, and still others present distinctly annular arrangement.

Subsequent examination of tissue removed from one of the circinate lesions was examined by Dr. Hamilton Montgomery and by Dr. Marcus R. Caro, each of whom felt that the histologic observations were consistent with the diagnosis of mycosis fungoides.

DISCUSSION

DR. UDO J. WILE, Ann Arbor, Mich.: The condition in this case does not resemble any that I have ever seen before, and the diagnosis is an interesting field for speculation. I wonder whether the answer may not lie in some of the fine petechial lesions which she had on her back. If these are, as it seems most likely, a part of the picture, they suggest some unusual dyscrasia of the blood. The nodular and serpiginous lesions on the shoulders and the hemorrhages could well be the results of leukemia. She has, however, no history of bleeding, no splenic enlargement and no enlargement of the superficial lymph glands. A careful examination of the blood might well throw some light on this unusual case.

DR. H. E. MICHELSON, Minneapolis: When a patient presents a lesion that begins in a fixed locality, remains there and progresses, it usually belongs to one of three groups: malignant growth, granuloma or lymphoblastoma. I believe that the microscopic study has ruled out both epithelioma and sarcoma. The lesions do not suggest any of the specific granulomas. I therefore believe that by exclusion the condition in this case falls in the lymphoblastoma group, and my guess is that it is of the mycosis fungoides type.

DR. EDWARD A. OLIVER: I saw this patient with Dr. Senear thjs morning. This was her first visit. I agree with Dr. Wile and Dr. Michelson that it is not possible to make a definite diagnosis without a histologic examination and a careful study of the blood; nevertheless, certain symptoms point strongly to mycosis fungoides, namely, the large tumor on the vulva, the annular configuration of several of the lesions on the neck and chest and the small mushroom-like tumor in the left groin. However, the absence of itching is not what one would expect in mycosis fungoides.

DR. FRANCIS E. SENEAR: As Dr. Oliver said, we feel that the condition belongs in the lymphoblastoma group. Because of the absence of a history of pruritus and because of the early lesions of eczematous type, one would think of mycosis fungoides of the d'emblée type. I do not think one can definitely say what the diagnosis is until a study of the blood and a biopsy have been made.

A Case for Diagnosis (Esthiomene?). Presented by DR. M. H. EBERT.

This 28 year old Negress, who was treated for syphilis in 1937, had a "wart" on the vulva which was never treated and never completely healed. About one year ago she noticed a gradually increasing painless swelling of the vulva. She has had no gastrointestinal complaints. She states that she never had a "bubo."

Examination of the blood revealed 54 per cent hemoglobin, 3,020,000 erythrocytes and 8,400 leukocytes, with a differential distribution of 84 per cent polymorphonuclear leukocytes, 13 per cent lymphocytes and 2 per cent eosinophils. The Wassermann reaction of the blood was 3 plus. The results of chemical examination of the blood and the urine were essentially normal. The reaction to a Frei test was 3 plus. No Donovan bodies were found on examination of the section removed for biopsy or in the smear.

DISCUSSION

DR. UDO J. WILE, Ann Arbor, Mich.: I discussed a number of years ago, with a group of gynecologists, the term esthiomene. This term is fast disappearing from medical nomenclature. In the older literature it appears to refer to painful ulcerated lesions in the vulva which frequently had to be examined under anesthesia because of the pain. The consensus seems to be that this term applied to granuloma inguinale and perhaps lymphogranuloma venereum, which were not recognized a decade or more ago. This particular case impresses me as a case of elephantiasis, such as one not uncommonly sees following infection.

DR. MAX S. WIEN: Esthiomene was the name first given by Huguier (Mémoire sur l'esthiomène ou dartre rongeante de la région vulvo-anale, *Mém. Acad. de méd.* **14**:501, 1848) to describe a chronic ulceration of the vulva associated with elephantiasis and sclerosis of the vulva. These conditions were subsequently described by Fournier in 1875 as "syphilome anorectal," but today it is known that in the majority of cases, this condition is associated with a positive Frei reaction and that the syndrome is produced by lymphogranuloma venereum. The clinical syndrome of lymphogranuloma venereum in females was classified by Ravaut, Seneque and Cachera into three types: (1) anorectal elephantiasis, (2) rectal stenosis and stricture and (3) pelvimetritis with secondary rectal stricture. The first stage in this sequence is esthiomene (ulcus vulvae chronicum elephantiasicum). Frei and Koppel (Ulcus vulvae chronicum elephantiasicum [Esthiomène] und sogenanntes Syphilóme anorectal als Folgeerscheinungen der

Lymphogranulomatosis inguinalis, *Klin. Wchnschr.* **7**:2331 (Dec. 2) 1928) called attention to the frequency of the occurrence of positive Frei reactions in cases of esthiomene. In addition there may be extension to the adjacent lymphatic vessels in the skin of the buttocks and perineum, with the production of localized soft granulomatous masses, some of which may ulcerate.

This case closely resembles some of the cases observed in the series studied by Dr. Perlstein and me at the Cook County Hospital (Wien, M. S., and Perlstein, M. O.: Lymphogranuloma Inguinale, *M. Rec.* **139**:288 [March 21] 1934), and the clinical picture is like that illustrated in figure 151 in Cabot's textbook (Wien, M. S.: Lymphogranuloma Inguinale, in Cabot, H.: Modern Urology, ed. 2, Philadelphia, Lea & Febiger, 1936, vol. 1, p. 235).

DR. MICHAEL H. EBERT: I had a particular purpose in presenting this case. At the recent meeting of the American Academy of Dermatology and Syphilology in the city of New York Dr. Cole conducted a clinic on venereal infections, including lymphogranuloma venereum and granuloma inguinale, and showed several excellent lantern slides. Among them were two that appeared to me to illustrate lymphogranuloma venereum, but he described them as illustrating hypertrophy or elephantiasis of the prepuce, due not to lymphogranuloma venereum but to granuloma inguinale.

When this woman came to the clinic I immediately thought she might have a double lesion, in spite of the fact that there was a positive Frei reaction. Biopsies were made and smears from the biopsy specimens and also various stains. Dr. Slepyan, who has done considerable work on the histology of granuloma inguinale, has examined the sections, and so have I. I have seen no Donovan bodies. There were some peculiarly stained mulberry-like bodies, which have to be studied further, but no Donovan bodies. So I go back to a diagnosis of elephantiasis of the vulva, particularly of the preputial area, of an extraordinary type on the basis of a lymphogranuloma venereum.

Steatocystoma Multiplex. Presented by DR. EDWARD A. OLIVER and DR. SAMUEL M. BLUEFARB (by invitation).

P. H., a Negro aged 31, is presented from the dermatologic clinic of Northwestern University with cystic lesions on his chest which have been present for the past four years.

On the sternal area of the chest there are about twenty cystic lesions, varying in size from that of a split pea to that of a coffee bean. The lesions are smooth and of the same hue as that of the surrounding skin. There are no subjective symptoms.

DISCUSSION

DR. CLARK W. FINNERUD: I have observed several cases of this kind since Dr. Ormsby and I reported some a few years ago, but I have never seen a Negro with this condition. I think that the disease is clinically distinctive and that the diagnosis can be made without even piercing one of the lesions and obtaining the characteristic content. The disease is characteristic histologically and is rather easily differentiated from the ordinary sebaceous cyst and from cystic abscesses of acne.

DR. OLIVER S. ORMSBY: I think it might be of interest to know that this patient had hundreds of lesions on the abdomen, thighs, chest and arms but none on the face. He had no sign of acne. A somewhat similar condition occasionally occurs in acne, but it is entirely different from that under discussion. Steatocystoma multiplex is not connected with acne in any way. When the contents of one of these cysts is removed, the cyst does not recur. Dr. Finnerud showed that the productive part of the cyst was destroyed, which prevented its reproduction. The fat is often an oily liquid like olive oil; it is occasionally solid. Dr. Ebert drained more than two thousand cysts in this patient, and the disease is now practically cleared up. Incision and removal of the contents of ordinary sebaceous cysts are usually followed by recurrence.

Psoriasis of the Palms and Soles. Pustular Psoriasis? Presented by Dr. Theodore Cornbleet and Dr. D. Cohen (by invitation).

T. A., a white man aged 72, has an eruption on the hands and feet which has been present a number of years but has become worse in the last year and a half. Now the eruption is mostly on the palms and soles, with a few lesions on the dorsal surfaces. The eruption consists of thick, scaly, sharply defined patches. The primary lesions appear to be flat pustule-like lesions, and these coalesce to form the larger ones. The roof of the primary lesions can be lifted off, exposing a bright red shiny floor with some punctate bleeding. There is no escaping fluid. Many lesions merely have limiting collaret scales. The nails have transverse striations, and some of the fingers show drumstick-like paronychial swellings. The usual sites for psoriasis are uninvolved.

DISCUSSION

Dr. James H. Mitchell: My impression is that the eruption is psoriasis rather than a pustular psoriasis of the palms and soles. Some of the lesions suggest pustules but actually are not. The lesions which one would expect to see are tiny superficial pustules, but none are present here.

Dr. M. H. Ebert: I agree with Dr. Mitchell.

Dr. Oliver S. Ormsby: The lesions of psoriasis limited to the palms present a different appearance from those seen in other areas. I had experience with a case that is worth recording. When first seen by me, a man had had the condition for a number of years, and it was still limited to the palms and soles. Later it gradually spread until it covered the entire cutaneous surface. Dr. Mitchell photographed that patient when he had but one small area of normal skin on the entire body. The condition then presented the appearance of a general exfoliative dermatitis, but it was a universal psoriasis. In the last stages epitheliomas developed on the forearms. These may have been on an arsenical basis. The work on arsenical epithelioma by Montgomery had not been done at this time. The interesting features were the limitation of the lesions to the palms and soles for years, later the generalization and the rebelliousness to treatment and finally the development of epitheliomas.

Dr. Theodore Cornbleet: The manifestations of pustular psoriasis I have seen, as Dr. Mitchell mentioned, were small pustules mostly on the thenar and hypothenar eminences, whereas in this case the lesions are much larger and mostly on the hollow of the palm. When the lesions are opened there is no content; the roofs overlie empty spaces and red bases. Psoriasis of the palm, just like that of the nails, is rather stubborn, according to my experience. The only treatment I have found of value is the use of roentgen rays. This man's palms were clear of lesions after four ¼ unit roentgen ray treatments, each given a week apart. Two weeks after the last treatment, however, the lesions recurred.

A Case for Diagnosis (Lymphoblastoma? Parapsoriasis?). Presented by Dr. Theodore Cornbleet and Dr. H. C. Schorr (by invitation).

M. B., a white boy aged 18, has an eruption which appeared as a single macule near the pubes one year ago. Since then new lesions have appeared while the old ones have remained. Now there are discrete macules, averaging in diameter about 1 inch (2.5 cm.). They are waxy red, of almost normal skin texture and fairly sharply outlined and remain discrete. There is no apparent scale. A couple of the lesions show telangiectases. There are no subjective symptoms. All the lesions are grouped about the lower part of the abdomen and groins.

A section from a lesion on the thigh showed the epidermis to be slightly corrugated. There was a thin scale, and the granular layer was discontinuous. The basal cell layer was intact, but there were a few lymphocytes scattered throughout the epidermis. In the subpapillary layer and extending into the papillae was a perivascular densely packed infiltrate composed largely of lympho-

cytes. Mitotic figures were not seen. Staining for iron showed a slight amount of iron pigment within histiocytes about these perivascular mantles.

DISCUSSION

DR. M. R. CARO: I could not make a definite histologic diagnosis in this case. I considered parapsoriasis as the most likely diagnosis. The slight changes in the epidermis, the invasion of the epidermis by a few cells and the presence of the infiltrate in the upper part of the corium speak for parapsoriasis. Against that diagnosis is the voluminous infiltrate present, more than is ordinarily seen. The profuseness of the infiltrate in the upper part of the corium suggests mycosis fungoides, but against this diagnosis is the almost uniform type of cell present.

DR. HAMILTON MONTGOMERY, Rochester, Minn.: This case is interesting because it brings up the relation of parapsoriasis to lymphoblastoma. Clinically, I believe the eruption would be accepted as typical of a parapsoriasis en plaques, although a few of the lesions are somewhat infiltrated and more sharply demarcated than is usual in parapsoriasis. Keil has stated that in practically all cases parapsoriasis en plaques represents an incipient stage of mycosis fungoides (ARCH. DERMAT. & SYPH. **38**:544 [Oct.] 1938). McCarthy (ARCH. DERMAT. & SYPH. **45**:81 [Jan.] 1942) has recently expressed the opposite view, that the common types of parapsoriasis remain as such and do not eventuate in mycosis fungoides or other lymphoblastomas. With Burkhart, at the Mayo Clinic, I recently completed a review of 52 cases of various types of parapsoriasis in which the diagnosis was confirmed by histologic studies; many of these have been followed for many years, and there has been no evidence of a transition to mycosis fungoides or other forms of lymphoblastoma. However, there were 6 additional cases in which the clinical diagnosis was parapsoriasis en plaques but the histologic examination showed evidence of mycosis fungoides and the disease eventuated as such. Dr. Ormsby in his book reported a case (observed also by Brocq) of a condition diagnosed as parapsoriasis en plaques which only after many years eventually developed into a lymphoblastoma. A number of similar cases are to be found in the literature.

In the case just presented, the infiltrate, as Dr. Caro has stated, is much greater than one sees in either parapsoriasis guttata or the en plaques type. A dense infiltrate even with necrosis may occur in parapsoriasis varioliformis of Habermann, which is probably an acute phase of parapsoriasis guttata. In the case under discussion, besides the dense infiltrate, there are pyknosis and karyorrhexis of the cells, multiplicity of the cell types and other features to suggest early mycosis fungoides. I should regard the condition in this case, therefore, as a premycotic stage of mycosis fungoides, in which the lesions clinically simulate those of parapsoriasis en plaques.

DR. OLIVER S. ORMSBY: I think that the relation of parapsoriasis to mycosis fungoides is clear. My experience does not corroborate Keil's view, that in all cases parapsoriasis en plaques is premycotic mycosis fungoides, though in many of them this is true. I am sure that Dr. Wile entertains the same opinion. Our patient, mentioned by Dr. Montgomery, who had parapsoriasis and then mycosis fungoides was under the care of Dr. Foerster for a long period. Dr. Brocq confirmed the clinical diagnosis of parapsoriasis in this case. Dr. Wise reported a similar case. These cases were reported from the clinical standpoint. I think there is no question about the fact that a premycotic mycosis fungoides with lesions of parapsoriasis may remain so for a long time before the development of the classic lesions. If any question exists it can be solved by an early biopsy, as was done in this patient. I believe that parapsoriasis en plaques is an entity that remains as such indefinitely.

DR. H. E. MICHELSON, Minneapolis: I was impressed with the fact that the lesions were all in the bathing trunk area and that they had a somewhat purpuric appearance. I do not feel that the condition is parapsoriasis. I should like to suggest purpuric lichenoid dermatitis as a possibility.

DR. F. W. LYNCH, St. Paul: Although the distribution of the eruption is atypical, my clinical impression is that the patient has parapsoriasis. However, after the microscopic examination, I changed my opinion because of the large amount of cellular infiltrate. I believe that this patient has some form of lymphoblastoma.

DR. UDO J. WILE, Ann Arbor, Mich.: I should like to differ with Dr. Michelson with regard to the localization of the lesions of parapsoriasis. In my experience these have a decided predilection for the groin, the upper part of the thigh and the lower portion of the trunk. This patient also, however, has lesions on his back. Their oval contour and peculiar brick red color are characteristic of the Brocq type of parapsoriasis. The relation of the en plaques form of parapsoriasis to mycosis fungoides is, I believe, an established fact. Undoubtedly, many conditions remain as parapsoriasis. The frequency, however, with which mycosis fungoides develops in cases of this type is striking. Years ago patients were shown before this group with a diagnosis of parapsoriasis and later were presented again, with mycosis fungoides.

DR. HAMILTON MONTGOMERY, Rochester, Minn.: I agree in the main with what Dr. Wile has just stated and that it may confuse the students for generalized erythroderma to be called mycosis fungoides. I wish to cite, however, a case of generalized erythroderma which was encountered at the clinic and regarded simply as one of eczema because the skin cleared up completely following hospitalization for a few weeks. The histologic picture was definitely that of early mycosis fungoides. Two years later the patient was seen by Dr. Barney in Cleveland, at which time classic clinical and also pathologic features of mycosis fungoides had just developed. Many of the other lymphoblastomas may also start out primarily as an erythroderma, and, conversely, patients presenting clinical characteristics of mycosis fungoides may prove histologically to have Hodgkin's disease or another of the lymphoblastomas. The persistence of the lesions without much progression for two years in Dr. Cornbleet's case is not inconsistent with the slow progress of the premycotic stage of mycosis fungoides. Correlation of the clinical and histologic observations and various laboratory findings in cases of the lymphoblastomas is important from the standpoint of prognosis. As a general working rule, when the histologic picture is that of mycosis fungoides without evidence of transition to lymphosarcoma and without any hemocytologic changes, the prognosis is much more favorable and the duration of the disease is likely to be longer than when evidence of multiple types of lymphoblastoma is to be found.

DR. THEODORE CORNBLEET: The histologic changes were characteristic of parapsoriasis en plaques. There were observations suggestive of lymphoblastoma. This man's lesions itch severely, which is not in keeping with parapsoriasis but suggests lymphoblastoma. In those subjects in whom parapsoriasis assumes the changes of lymphoblastoma, does pruritus become prominent after being absent?

DR. OLIVER S. ORMSBY: In the case I observed in which parapsoriasis en plaques eventually developed into mycosis fungoides, itching was present. Looking back over all the cases published as instances of parapsoriasis that had gone into mycosis fungoides, I find that there usually was itching. This is not important, however, as itching may be present or absent in both conditions, though it is usually absent in parapsoriasis and present in mycosis fungoides.

A Case for Diagnosis (Multiple Sebaceous Adenoma?). Presented by DR. THEODORE CORNBLEET and DR. H. C. SCHORR (by invitation).

F. M., a white man aged 62, has had an eruption on the face for forty years. The lesions are noninflammatory nodules that have the average size of a split pea. They are smooth, lenticular, sharply outlined, pinkish or skin colored and translucent. A few are umbilicated, and some of them coalesce. The bulk of the lesions are on the middle of the forehead with a few on the cheeks. There are no subjective symptoms.

A section from a lesion on the forehead shows a normal epidermis, which is slightly flattened. Throughout the corium there are many hypertrophic but otherwise normal sebaceous glands.

DISCUSSION

DR. RUBEN NOMLAND, Iowa City: Clinically these lesions are typical hypertrophic sebaceous glands, such as are seen in senile persons. I suppose they are adenomas of the sebaceous gland that occurred at puberty or before rather than waiting until later life to occur.

DR. UDO J. WILE, Ann Arbor, Mich.: I have observed many cases similar to this and found that the lesions microscopically are not sebaceous but are localized examples of colloid degeneration.

DR. M. R. CARO: These lesions histologically are hypertrophic sebaceous glands, the sections being identical with those described by Dr. Nomland.

DR. THEODORE CORNBLEET: My impression was that the lesion described by Dr. Nomland occurred later in life. Those present in this case interest me because they occurred so early in life.

Blastomycosis. Presented by DR. EDWARD A. OLIVER and DR. M. A. GOLD (by invitation).

J. J., a Negro aged 49, states that on Feb. 8, 1940 he noticed a small pinhead-sized ulcer on the left upper eyelid. This spread gradually and became larger. Two weeks later he noticed a similar ulcer on the left cheek near the eye. The lesions grew considerably larger, and he went to the Veterans Administration Facility in Memphis, Tenn., where he was given roentgen ray therapy. The lesions continued to spread, and he was admitted to the Veterans Administration Facility at Hines, Ill., on Dec. 23, 1941. Examination showed a rather typical involvement of the skin about the left orbital region and the eyelids, a blastomycetic dermatitis.

Wassermann and Kahn reactions were negative. The urine and the blood count were normal. The reaction to an intradermal test with blastomycin was 4 plus. A roentgenogram of the chest showed the pulmonary fields to be clear.

Histologic examination of tissue from an ulcer of the lower left eyelid revealed numerous intradermal abscesses. Within these abscesses there was a foreign body type of reaction with epithelioid cells and an occasional foreign body giant cell. Numerous double-contoured organisms were noted, and in some instances budding of the organisms was seen.

DISCUSSION

DR. H. M. HEDGE: I had a case like this at Northwestern University; the lesions resembled closely those around the eye and on the forehead in this case. My patient had two lesions on the buttocks and two on the forehead. This patient had a lesion on the hand. I removed them with the electrocautery, and they did not return. A few of the small lesions were frozen by solid carbon dioxide, and they remained away. The lesions around the eye were treated by cautery. The patient had been given iodides and also some ultraviolet irradiation. The electrocautery, however, with the internal use of iodides, I think, actually did more than anything else to bring about a cure.

DR. HARRY R. FOERSTER, Milwaukee: I have seen lesions of blastomycosis on the eyelid, and they are difficult to treat. The great amount of destruction of the eyelid in this case indicates the importance of early recognition and early treatment in such cases. I believe my best results followed treatment with intravenous injections of sodium iodide, in addition to whatever local treatment was indicated in the individual case. For an early lesion of the eyelid I favor the use of moderately filtered radium, with a radium plaque, in full dosage. In this case I do not believe that much can be done in the way of local treatment to save the lid on account of the destruction that has taken place, but intensive intravenous treatment may stop the process.

DR. RUBEN NOMLAND, Iowa City: In the past two years I have had the opportunity of seeing 2 patients with extensive involvement of the eyelids, almost as severe as this man's. Both patients recovered under intensive internal treatment with sodium iodide. For 1 of them it was necessary to do considerable plastic work after recovery, but the amount of scarring was slight, considering the severity of the process. The interesting feature in blastomycosis of the eyelids is that it involves the skin only. In the cases I have observed there was no involvement of the globe. Why the globe is immune to blastomycosis that comes in its immediate vicinity is something I do not understand.

DR. LOUIS A. BRUNSTING, Rochester, Minn.: Blastomycosis cutis is an interesting disease, and at present it seems to be in a cycle of relative rarity. For the most part, it remains an isolated cutaneous type of infection; only occasionally does it become widely disseminated. In the localized form there is an apparent immunity of the healed sites to reinoculation, whereas the adjacent normal skin offers small resistance to the progressive process. By the combination of systemic treatment with large doses of iodides and by the application of sizable doses of filtered radium to the sites the condition can be controlled in most instances, but occasionally resistant examples of the disease extend over a period of years.

DR. EDWARD A. OLIVER: I presented this case merely because I thought that the number of cases observed around Chicago is becoming smaller every year.

I can answer Dr. Brunsting's question by saying that at the Veterans Administration Facility, where the patients come from all over this part of the country, there were 8 cases of blastomycosis in the last year.

I am glad to have the therapeutic suggestions because I have a man in the hospital now with a refractory lesion on the lower eyelid which I have hesitated to treat surgically. He has had large doses of sodium iodide intravenously and potassium iodide by mouth. No radium has been applied, and no operation has been performed. The patients with this disease have been given large doses of potassium iodide by mouth, intravenous injections of sodium iodide and copper sulfate and iontophoresis locally, with good results.

DR. OLIVER S. ORMSBY: Dr. Brunsting asked a question concerning immunity which Dr. Oliver did not answer. In regard to local immunity, I have seen foci develop a number of times in scar tissue in healed areas. Usually, however, the extension is into new territory. I have seen the disease on the lids in a number of cases. The organism was described originally by Gilchrist in 1894 (*Bull. Johns Hopkins Hosp.* **1**:269, 1896). He showed sections containing the organism of blastomycosis before the American Dermatological Association, taken from a case of Dr. Duhring's which he had considered to be one of scrofuloderma. A large number of cases were observed and studied in the Chicago district for several years following 1898. At one time during this period cultures were obtained from about 100 different cases The number remained comparatively high for several years and then declined. The number of cases of both cutaneous and systemic blastomycosis is relatively small at present. The disease is relatively rare in Europe, where early investigators thought it was a yeast infection implanted on syphilis or tuberculosis.

As to treatment, in most of my cases the disease responded to iodides. In some it was extensive and required long-continued treatment in large dosage. In cases in which it became resistant to iodides this treatment was temporarily suspended and a series of about five injections of arsphenamine given, after which administration of iodides was resumed and gave good results. It seems that arsphenamine sensitizes the organism of blastomycosis to iodides, and when use of iodides is resumed the condition clears up. Recently a vaccine has been used with success for persons with iodide-resistant blastomycosis.

DR. DAVID LIEBERTHAL: From a historical point of view, I should like to mention that one of the first cases of blastomycosis published was that of Drs. Hyde, Hektoen and Bevan. Another case was then reported before the Chicago

Medical Society in May 1899 by Dr. Daniel Eisendrath; he presented illustrations and pathologic specimens. In the discussion I suggested that in these cases, from a clinical standpoint, one might be dealing with blastomycosis implanted on either tuberculous or syphilitic lesions. Dr. Bevan, who was also present at this meeting, agreed with my remarks and said that the condition in his case (just mentioned), which he suspected of being a gummatous lesion, had yielded to the administration of potassium iodide. This preparation has since been a valuable remedy in this disease. In January 1900 a paper by Drs. Anthony and Herzog was published (Anthony, H. G., and Herzog, M.: A Case of Blastomycetic Dermatitis Engrafted on Syphilitic Ulcers, *J. Cutan. & Genito-Urin. Dis.* **18**:1, 1900) in which a full account of this discussion at the Chicago Medical Society was given.

Lymphoblastoma. Presented by Dr. Edward A. Oliver and Dr. M. A. Gold (by invitation).

J. M., a white man aged 53, stated that in January 1940 he noticed some small red spots which he thought were mosquito bites around his ankle. The lesions were pruritic, spread rapidly and coalesced until the eruption became generalized. The skin of the entire body became red and pruritic, and there was considerable scaling.

In June 1940 he was admitted to the United States Marine Hospital for treatment of this condition. He left there in October 1940 and spent some time in the University Hospital at Ann Arbor, Mich., where it was discovered that the liver was enlarged. The cutaneous picture was much like it is today. A biopsy of skin from the back was reported as showing a heavy infiltration of lymphocytes in the subpapillary region of the corium. The infiltration extended higher than usual into the papillary layer. After a short stay in the University Hospital, he was readmitted to the United States Marine Hospital in Detroit. He stayed there a short time and then went to the tumor clinic at the United States Marine Hospital in Baltimore. On Jan. 6, 1942 he was sent to the Veterans Administration Facility at Hines, Ill., for further treatment. Examination at that time showed a generalized erythroderma with adherent scales covering the entire body and a decided enlargement of the inguinal and axillary glands. The liver and spleen were both enlarged and palpable.

The Wassermann and Kahn reactions were negative. The urine was normal. Examination of the blood on January 12 showed: hemoglobin 75 per cent, erythrocytes 4,080,000, platelets 121,000 and leukocytes 13,700, with a differential distribution of 30 per cent polymorphonuclear leukocytes, 66 per cent lymphocytes (10 per cent young forms), 3 per cent monocytes and 1 per cent lymphoblasts. On January 15 examination of the blood showed 22,600 leukocytes, with a differential count of 45 per cent polymorphonuclears, 2 per cent monocytes, 50 per cent lymphoblasts (12 per cent young forms), 1 per cent basophils and 2 per cent metamyelocytes.

An inguinal gland was removed for histologic examination. It was 6 cm. in diameter, gray, homogeneous and soft. Microscopic examination revealed an infiltration of small round cells throughout, partly obliterating the architecture; there were many lymphoid follicles remaining, however. There was some proliferation of the reticulum. In some areas there was an attempt at fibrosis, and in these areas there were numerous eosinophils. There were no Sternberg-Reed cells. The capsule was invaded. This new growth was definitely a lymphoblastoma and in some areas showed some characteristics of Hodgkin's disease, although not sufficient for a definite diagnosis. The diagnosis was lymphoblastoma.

Microscopic examination of the skin revealed diffuse infiltration just under the epithelium. The infiltrating cells were nearly all lymphocytes, although there were numerous mononuclear leukocytes and a few atypical cells which could not be placed in any particular category. In other areas the cells surrounded the hair follicles and sweat gland structures; this was undoubtedly a lymphoblastic infiltration.

DISCUSSION

DR. HAMILTON MONTGOMERY, Rochester, Minn.: The histologic picture is typical of mycosis fungoides, making this condition a generalized erythroderma belonging in the group of lymphoblastomas.

DR. UDO J. WILE, Ann Arbor, Mich.: I had the opportunity of seeing this patient at the University Hospital some years ago. I believe he presents an excellent example of a disseminated scaly erythroderma which is lymphoblastomatous. I believe that one should restrict the term mycosis fungoides to a distinct clinical entity rather than apply it to a pathologic one. Mycosis fungoides, while commonly enough characterized by polymorphous infiltrate, may present any one of a number of different pathologic pictures. I have seen clinical mycosis fungoides characterized pathologically by lymphosarcoma, by Hodgkin's disease, by leukemia and, in at least 2 cases, by pure eosinophilic tumors. For this reason, to diagnose a scaly erythroderma as mycosis fungoides from the pathologic picture is to my mind confusing a pathologic picture with a clear clinical syndrome.

DR. EDWARD A. OLIVER: I believe this patient shows many of the signs of leukemia of the skin. I hesitate to disagree with Dr. Montgomery, but several other pathologists have seen these sections and have made a diagnosis of leukemia. The generalized erythroderma has been present for two years. I believe that by this time if the condition were mycosis fungoides the patient would have an entirely different group of symptoms. My conception of mycosis fungoides is much different from the picture this man presents.

So-Called Myoblastoma of the Skin. Presented by DR. FREDERICK R. SCHMIDT.

Histologic sections of nodules removed from the skin of 3 patients are presented. The average diameter of the nodules was 8 mm.

The microscopic pictures of the nodules in the skin are similar; hence they will be described together. Large, oval, elongated and polygonal cells, single and in sheets, together with syncytial-like multinucleated strands are seen between bundles of connective tissue in the papillary and reticular zones of the corium. The cytoplasm is abundant and has a vacuolated and coarsely granular structure. No evidence of the arrangement of the granules into rows suggestive of cross striations is noted. Most of the nuclei are small, round or oval. The granules in frozen sections did not stain with sudan III or nile blue sulfate. The granules are well seen in sections stained with Mallory's phosphotungstic acid–hematoxylin stain.

DISCUSSION

DR. HAMILTON MONTGOMERY, Rochester, Minn.: This condition is a definite and distinct entity. When Dr. Schmidt sent me sections from these cases a year ago I missed the diagnosis, although I suspected there might be degenerating muscle of some sort. At the last meeting of the American Academy of Dermatology and Syphilology, in December 1941, Dr. Machacek presented 3 cases of myoblastoma at the dermatopathologic round table. This disease occurs most commonly in the striated muscle of any organ but especially in that of the tongue, where it can develop into rhabdomyoma or rhabdomyosarcoma. Involvement of the skin is relatively rare. The cutaneous lesions may be solitary or multiple and resemble dermatofibroma (histiocytoma). The characteristic cell is a large foam or xanthoma-like cell containing no lipids but containing numerous fine granules the nature of which remains to be determined. These cells apparently rise from striated muscle and are regarded either as degenerating muscle cells or, by most authors, as the embryonic state of striated muscle. When the lesions are found on the skin or mucous membranes, there is associated frequently a pseudoepitheliomatous hyperplasia of the epidermis. Thus, in the past this lesion has been described as squamous cell epithelioma with myxomatous or sarcomatous degeneration of the connective tissue beneath the tumor. Myoblastomas may increase in size but are only locally malignant, according to existing knowledge of the cutaneous lesions of this condition.

DR. FREDERICK R. SCHMIDT: I presented these sections two years ago as illustrating sweat gland adenoma. Dr. Wile said emphatically that the lesion was not a sweat gland adenoma. Then a second case appeared, and Dr. Tuta and I thought of leiomyoma, showing degenerative changes. We went through a whole galaxy of diagnoses, including histoplasmosis of Darling, until one day this summer while looking over the general pathologic literature we saw a picture exactly similar to that in our sections. These sections are from solitary tumors, one each removed from 3 patients. Studies are now being conducted to find out what these granulations are. So far no evidence has been found that these tumors contain glycogen. I do not believe that they are derived from myeloblasts or other muscle-building elements. If they do come from muscle elements one has to assume, on the basis of a dysontogenetic theory, that there is striated muscle present in the skin.

LOS ANGELES DERMATOLOGICAL SOCIETY

KENDAL FROST, M.D., *President*

ARTHUR FLETCHER HALL JR., M.D., *Secretary*

Oct. 14, 1941

Keratosis Palmaris et Plantaris (Congenital). Presented by DR. SAMUEL AYRES JR.

S. B., a white boy aged 11 years, presents hyperkeratosis of the palms and soles, consisting of thickened yellowish areas, with a rather sharp line of demarcation along the sides of the feet and hands. These changes first appeared when the patient was about 6 weeks old. The father, one of eleven children, is the only member of the family known to suffer from a similar condition.

DISCUSSION

DR. L. F. X. WILHELM: I agree with the diagnosis. I have nothing to offer as to therapeutic possibilities.

DR. SAMUEL AYRES JR.: I presented this case for the purpose of hearing discussion as to whether a patient with this disease might be benefited by vitamin A therapy in view of the changes reported in some cases of ichthyosis and in Darier's disease.

Necrobiosis Lipoidica Diabeticorum. Presented by DR. SAMUEL AYRES JR.

O. D., a white woman aged 37, first noticed a lesion on her leg three years ago, which began as a small area resembling a cigaret burn and has gradually spread. On the anterolateral aspect of the right leg there is an oval lesion, about the size of a dollar, with a sharply defined, slightly elevated, almost pearly-looking border and a slightly atrophic center with dilated capillaries. The entire lesion has a somewhat transparent appearance, with a yellowish to violaceous color. Near the center is a pea-sized, slightly crusted area. The result of a dextrose tolerance test and the cholesterol level of the blood (150.6 mg. per hundred cubic centimeters) were not suggestive of diabetes mellitus, but histologic changes in the lesion were characteristic of the diagnosis as presented.

Late Syphilis with Arsenical Dermatitis and Infectious Relapse Under Treatment. Presented by DR. BEN A. NEWMAN.

E. C., a white woman aged 58, gives no history of early syphilis. The first lesions, noticed in 1937, consisted of a group of granulomatous ulcers on the left forearm, which were treated as tuberculosis cutis because the histologic diagnosis

was tuberculous granuloma and she reacted positively to old tuberculin in a dilution of 1:1,000,000; a single Wassermann reaction was reported as negative at that time.

In September 1939 the persisting lesions on the forearm were thought to be gummas, and Kahn and Kline reactions of the blood were positive. The lesions healed promptly under treatment with neoarsphenamine, leaving the atrophic scars which are now present.

After she had tolerated ten injections of neoarsphenamine and twelve injections of bismuth subsalicylate, a generalized exfoliative dermatitis developed after one injection of mapharsen, 0.04 Gm. The administration of bismuth subsalicylate, given during the attack of exfoliative dermatitis, had to be discontinued after the sixth injection because of gingivitis; within two weeks there developed temporal alopecia, weakness, loss of weight, split papules at the angles of the mouth and moist papules on and around the genitals, showing Spirochaeta pallida on dark field examination, and a dime-sized annular infiltrated lesion on the cheek. All relapsing lesions cleared promptly after six daily injections of mercuric succinimide. Administration of this drug was continued in doses of ⅓ grain (0.02 Gm.) twice weekly for eight months, at the end of which time the Wassermann and Kahn reactions were negative. After a six months' rest period and three more months of mercury therapy the Wassermann reaction was still negative, and she has received only potassium iodide since that time (ten weeks ago). The patient returns tonight with a large bean-sized granulomatous ulcerated nodule with serosanguinous discharge on the left side of the scalp, apparently a gummatous relapse. The spinal fluid is normal, and there is no serologic or clinical evidence of syphilis in the husband.

DISCUSSION

DR. C. R. ANDERSON: I had only one thought: Malarial or typhoid fever therapy is indicated.

DR. BEN A. NEWMAN: This case presents a number of remarkable features: (1) the presence of a noduloulcerative gumma simulating, and treated as, tuberculosis cutis for two years, before antisyphilitic therapy was started; (2) the development of a severe arsenical dermatitis following a single dose of mapharsen, after an entire course of neoarsphenamine and of bismuth subsalicylate injections had been tolerated; (3) the development while she was receiving bismuth of infectious syphilids containing S. pallida, as well as other features of early syphilis; (4) the excellent clinical and serologic response to treatment with mercury; (5) the appearance of the present noduloulcerative gumma after a short rest period, in spite of absence of serologic evidence of syphilis. In brief, this patient has late cutaneous syphilis, and, after the usual satisfactory therapeutic response was obtained, an arsenical dermatitis developed, thereby destroying the defensive mechanism of the body so that a relapse of an early infectious type of syphilis occurred. Then, after an apparent therapeutic response, both clinically and serologically, another lesion of late cutaneous syphilis appeared. Undoubtedly, the type of treatment required for the future is one that will enhance the defense mechanisms of the patient.

DR. F. E. CORMIA (by invitation), Montreal, Canada: I think that a patient who has reacted as vigorously to mapharsen might not tolerate old arsenic. I agree with Dr. Anderson that fever therapy followed by the administration of a heavy metal might offer the best possibilities.

DR. WILLIAM GOECKERMAN: The negative serologic reactions do not speak against the diagnosis. Nor is it entirely impossible that the reactions may become positive again. But, on the whole, in such a case there should be a good prognosis.

Rosacea-Like Tuberculid of Lewandowsky. Presented by DR. BEN A. NEWMAN.

E. M., a white woman aged 38, has been in good health in spite of having had hypertension during the past year and mitral stenosis since childhood. Her erup-

tion first appeared on each cheek five months ago and has gradually spread to involve the present areas. She now shows a reddish eruption involving both cheeks, the chin and to a lesser extent the nose. Palm-sized patches on the cheeks extend from the nasolabial fold about two thirds of the way to the angle of the mandible and consist of grouped pinhead-sized or larger papules, varying in color from red to brown. The papules are aggregated; some are discrete, but the majority are confluent. On diascopic pressure they present an apple jelly color. There are many telangiectatic vessels throughout the involved area, with the telangiectasia involving many of the individual papules. The results of blood counts and urinalyses were normal. A roentgenogram of the chest was normal except for showing enlarged hilar glands. The reaction to a tuberculin test with purified protein derivative, first strength, was negative, but the reaction was positive to old tuberculin in a dilution of 1:5,000. Histologic examination showed tuberculous architecture consisting of epithelioid cells and lymphocytes in the corium. She received treatment with gold sodium thiosulfate for one month without improvement, but there has been decided improvement since the positive tuberculin reaction one week ago.

DISCUSSION

DR. CHRIS HALLORAN: I think this case is a typical one of rosacea-like tuberculid of Lewandowsky.

DR. F. E. CORMIA (by invitation), Montreal, Canada: I was wondering about the regional distribution of these cases. One never encounters a case like this in Montreal.

DR. KENDAL FROST: Geographic distribution of cases of tuberculid affords much ground for discussion. One rarely sees lupus vulgaris here, and yet at the Los Angeles County Hospital there is a fair number of instances of tuberculid. This is the second case of this disease that I have encountered here. In this one, I understand, the tuberculid developed here. The other one, which was presented by Dr. Saul Robinson, was that of a patient who had moved here from New York, where she lived when the eruption developed.

DR. BEN A. NEWMAN: This patient was presented because hers is the most classic rosacea-like tuberculid I have seen. An interesting feature is her failure to react to purified protein derivative. When she was retested with a 1:5,000 solution of old tuberculin two weeks ago the reaction was so intense that it still persists. Coincidental with this reaction to old tuberculin, there has been a decided improvement in the facial eruption.

Lupus Erythematosus with Elephantiasis of the Lip. Presented by DR. BEN A. NEWMAN.

H. B., a white youth of 19, has suffered from recurrent swelling of the lower lip since he received a severe sunburn one year ago. There have been persistent dermatitis and swelling of the lip for the past three months. The edema and erythema of the lower lip now involve the mucous surface and vermilion border, while a dry erythematosquamous patch extends 5 mm. onto the margin of the skin.

DISCUSSION

DR. WILLIAM GOECKERMAN: I have not been able to raise much enthusiasm about vitamins, but I should like to see riboflavin tried in this case, in 5 mg. doses twice a day. I think the disorder suggests ariboflavinosis. Most investigators today believe that ariboflavinosis is always associated with deficiency of other vitamins.

DR. C. R. ANDERSON: I think the diagnosis is lupus erythematosus.

DR. SAMUEL AYRES JR.: The patient has an interesting lesion on one sole. The lesion on the sole looks to me like lupus erythematosus. I have never seen this disease on the sole before, but it certainly has that appearance. Referring again to the question of riboflavin deficiency, which must be considered here

because of the cracking and fissuring of the mouth, I wonder if any one has ever shown any relation between riboflavin deficiency and a monilia infection, such as perlèche. Is perlèche attributable to riboflavin deficiency?

DR. SAUL ROBINSON: I suggest the diagnosis of recurrent erysipeloid. I should like to see sulfathiazole tried. The condition appears to be recurring from time to time.

DR. ANKER JENSEN: I have seen this man once before, and at that time I thought he was suffering from avitaminosis. The only lesions he presented then were fissuring and desquamation at the commissures of the lips. There was also a slight amount of swelling of the lower lip, which I thought was secondary.

DR. BEN A. NEWMAN: Clinically three possibilities exist: (1) contact dermatitis, (2) psoriasis and (3) lupus erythematosus. The elimination of dentrifices and all possible contact substances without improvement and histologically the failure to demonstrate intercellular or intracellular edema of the epidermis rule out contact dermatitis. Psoriasis can be ruled out by the absence of any of the histologic features. The history of the onset and exacerbation following sunburn, the histologic picture of hyperkeratosis, an otherwise normal epidermis and strands of lymphocytes in the upper half of the cutis and the slight tinctorial changes favor the diagnosis of lupus erythematosus. The patient states that he has been taking moderate doses of vitamin B complex for two months. I shall administer riboflavin as well.

A Case for Diagnosis (Lupus Erythematosus?). Presented by DR. HARRY P. JACOBSON.

I. C., a Mexican woman aged 32, states that her present eruption appeared six weeks ago, six weeks after a severe sunburn. She shows an extensive, roughly symmetric eruption involving both sides of the face, nose and forehead. The eruption on the flush areas of the face is made up of discrete and grouped vesicular elements, varying in size and consistency, situated on erythematous and edematous skin. The bridge of the nose is involved in a purely erythematous eruption, presenting no vesicular elements. The vesicular elements were of different stages of evolution. Her temperature by mouth this afternoon was 99.5 F. Her hemogram was normal except for a white blood cell count of 10,000, with 2 to 6 per cent eosinophils. The skin has shown some improvement after sulfathiazole (2-[para-aminobenzenesulfonamido]-thiazole) therapy during the past week.

DISCUSSION

DR. SAMUEL AYRES JR.: It does appear that the patient may have a contact dermatitis. According to the appearance of her face tonight, she might be sensitive to cosmetics or some other substance.

DR. SAUL ROBINSON: The patient has an atypical type of seborrheic dermatitis, It may be that the condition on the cheeks is of the same nature. Why not try a iittle salicylic acid and sulfur ointment?

Urticaria Pigmentosa Acquisita. Presented by DR. C. R. ANDERSON.

M. J. K., a white woman aged 33, states that about five years ago small brown spots began to appear on the legs and back. Some of the lesions were pink and pruritic at first but later became flat and pale brown. More recently new lesions have appeared on the face and ankle. There are now many pale brown, pea-sized to split pea–sized macules symmetrically disseminated over the face, back, chest, legs and ankles. Many of these lesions on the ankle have a pinkish cast. The individual lesions urticate on stroking. In addition, there are numerous café au lait spots and pigmented nevi scattered over the body. Histopathologic examination showed in the upper parts of the cutis numerous mast cells, well demonstrated by Unna's polychrome methylene blue stain.

Keratosis Pilaris, Generalized. Presented by Dr. Ben A. Newman.

H. B., a white youth aged 19, has had the present rough, dry skin since infancy, and he had recurring infantile eczema of the cheeks until he was 8 or 9 years of age. He shows a generalized papular, follicular, noninflammatory eruption involving the trunk and extremities. The lesions are like those of exaggerated and horny goose flesh. Histologic examination showed follicular hyperkeratosis.

DISCUSSION

Dr. Saul Robinson: This patient told me that his eruption had been present since birth. I thought it was a form of ichthyosis. Many of the lesions look like lichen pilaris. I should be interested to know the result of intensive vitamin A therapy.

Dr. Nelson Paul Anderson: I think this case is most interesting. I doubt that the patient has a vitamin A deficiency. I questioned him closely as to his ingestion of carrots and found that he eats great quantities of them raw. I think if Dr. Newman will examine his patient's palms closely he will find a definite yellowish tint. If this condition is a vitamin A deficiency, it must be due to improper utilization rather than to inadequate intake.

Dr. Ben A. Newman: About ten years ago this eruption was called nevoid dermatitis by Dr. Moses Schultz. However, during the past two years follicular keratoses of this type in localized forms, have been seen to respond to large doses of vitamin A. Chemical determinations for vitamin A are now being made, and when these studies are completed this patient will be given vitamin A in large doses. If this eruption should undergo involution with the administration of vitamin A, in spite of the fact that the patient has a carotenemia, it would lead to a better understanding of the mode of action of this vitamin.

A Case for Diagnosis (Chromoblastomycosis?). Presented by Dr. Nelson Paul Anderson.

J. B. P., a white man about 40, born in Brazil, has had an itching eczematous and pustular eruption involving the legs for the past six or seven years. He now shows a large eczematous patch on the outer anterior aspect of the right leg, ulcerating, crusted and granulomatous lesions on the medial aspect of the right leg and a larger than dollar-sized thickened lichenified patch on the dorsum of the left foot. He also has a few eczematous and intertriginous lesions behind the right ear and on the penis. Both large toe nails are undermined and deformed. The Kahn and Kline reactions were both strongly positive. Cultures of material from the lesion on the leg showed Staphylococcus aureus, and the toe nails yielded Aspergillus on two occasions. Biopsy showed only infected granulomatous tissue. Bismuth by mouth and potassium iodide by mouth brought about no change in the lesions.

DISCUSSION

Dr. Arthur Fletcher Hall Jr., Santa Monica, Calif.: I suggest the possibility of a Senear-Usher type of pemphigus. The lesion on the dorsal aspect of the left foot suggests lupus erythematosus. The patient states that a great many lesions appear as vesicles and gradually turn into the type of plaque that is seen on the right shin.

Dr. William Goeckerman: It is at once apparent that this eruption is not a common one. Chromoblastomycosis is a rara avis in my experience. I recall having encountered only 1 case.

Dr. Nelson Paul Anderson: The lesions looked like eczema at first. In the history, however, there are several pertinent facts. The patient was born in São Paulo, Brazil, and lived there until he was 8 years of age. São Paulo is the fountainhead of chromoblastomycosis, so far as the reported cases are concerned.

He has a positive Wassermann reaction. He has had one year of thorough antisyphilitic treatment. His blood was normal in March or April, and he had a negative Wassermann reaction a year previous to that time. The lesions developed during his antisyphilitic therapy.

Chromoblastomycosis entered my mind because the patient came from São Paulo, Brazil. The eruption is certainly not an ordinary eczema. The last case of chromoblastomycosis reported in this country was that of a Negress from Philadelphia and was reported by Weidman and Rosenthal (ARCH. DERMAT. & SYPH. **43**:62 [Jan.] 1941). Eight years elapsed before a diagnosis of chromoblastomycosis was made. The woman also had syphilis. At biopsy the organisms were found in the sections, and only then was the correct diagnosis made. The organisms were never recovered by culture. I do not know whether the diagnosis is chromoblastomycosis in this case but suggest that possibility.

Psoriasis, Recalcitrant to Treatment. Presented by DR. NELSON PAUL ANDERSON and DR. SAMUEL AYRES JR.

S. H., a white woman about 50, was seen first on Oct. 14, 1940, at which time the eruption had been present a year. It had resisted various types of treatment, including application of proprietary ointments, iodine, ammoniated mercury and coal tar. The eruption is seen mostly on the anterior portions of the legs; it consists of grouped, bright red, slightly scaly plaques and covers an area approximately the size of a hand. There are several smaller papules, pea-sized, covered with silvery scales and a similar lesion on the left elbow. Treatment has consisted of fifteen fractional doses of roentgen rays, ultraviolet irradiation, calcium thiosulfate intravenously, autohemotherapy and application of many ointments, including naftalan, chrysarobin, chlorhydroxyquinoline (quinolor), juniper tar and coal tar ointments as well as wet dressings of solution of aluminum acetate, 0. 25 per cent silver nitrate solution and various types of bland applications. The patient has also applied a patent remedy known as siroil (according to a recent label, siroil contains not more than 0.4 per cent of mercuric oleate and "also cresol, vegetable oils, mineral oils and inert ingredients in varying amounts"). None of these preparations has been of any particular benefit. There was one period when the eruption almost cleared up, but it promptly recurred. Microscopic examination of scrapings showed no fungi. The patient is presented for therapeutic suggestions.

DISCUSSION

DR. SAUL ROBINSON: I agree with the diagnosis. In some cases this disease is resistant to therapy. I wonder whether there is any element of stasis. I think a biopsy should be made.

DR. F. E. CORMIA (by invitation), Montreal, Canada: I think Dr. Robinson's suggestion is excellent. I have had cases in which I could not clear the psoriatic lesions without stasis support.

DR. ANKER JENSEN: Some persistent psoriatic lesions when frozen with ethyl chloride will disappear. However, I should use this method only as a last resort.

DR. ARTHUR FLETCHER HALL JR., Santa Monica, Calif.: I tried freezing with ethyl chloride in several cases about a year ago, and, although some lesions improved greatly, I did not feel that the results were particularly satisfactory. I suggest treatment with vitamin P (citrin lemonade). I have used it in several cases of resistant psoriasis, and in a fairly large percentage the disease has improved. Two patients with psoriasis of the scalp were willing to have other treatment withheld, and almost complete involution of the lesions followed the use of vitamin P. In 1 case of psoriasis of the palms and soles which was extremely resistant to treatment of any kind, with the administration of citrin lemonade the lesions on the palms were kept under control and those on the soles cleared com-

pletely. The patient can prepare the citrin lemonade herself. I think this therapy is at least worth a trial.

DR. SAUL ROBINSON: Dr. Lawrence has been using vitamin P at the Los Angeles County Hospital for the treatment of psoriasis. I should like to hear his conclusions as to the value of this treatment.

DR. HERBERT LAWRENCE (by invitation): In 1 case this treatment was a failure. The other 3 or 4 cases I am not in position to report on now.

Hemangioma. Presented by DR. JOHN D. ROGERS (by invitation).

K. S., a white man aged 27, presents a soft, tumor-like lesion, showing definite pulsation, on the bridge of the nose and somewhat to the left. In the center there is what appears to be a bluish spot, composed of vessels. The lesion has been present for ten years, with moderate increase in size during this time. A roentgenogram shows the structure of the nasal bones to be somewhat altered, there being a decreased osseous density and coarseness but no distinct localized lesion of the bones. An undetermined amount of radium treatment has been given, without improvement.

DISCUSSION

DR. NELSON PAUL ANDERSON: I think this lesion is a cirsoid aneurysm. The patient fell and injured his nose, after which there developed an angiomatous process. I think this traumatic angioma might remain as it is, but such lesions have been known to spread rapidly throughout the scalp and require extensive surgical measures. I suggest surgical intervention now.

DR. ANKER JENSEN: If this condition is a simple hemangioma I believe the method of choice for the treatment is injection.

DR. MAXIMILIAN OBERMAYER (by invitation): Administration of a sclerosing agent may be fatal.

DR. SAUL ROBINSON: I should consider the possibility of lymphangioma. The lymphatic element might be more important than the angiomatous part. The lesion is soft to the touch and unlike an angioma.

DR. JOHN ROGERS (by invitation): The first diagnosis I made was hemangioma. After the patient had had radium treatment, I examined the lesion closely and felt a definite pulsation. I am convinced that there is an artery involved.

A Case for Diagnosis (Reticulated Atrophy?). Presented by DR. NELSON PAUL ANDERSON and DR. SAMUEL AYRES JR.

M. S., a white woman aged 37, states that for the past six or seven years she has had an eruption on the face which seems to become worse when she is nervous or if she has an upset stomach. She presents a peculiar diffuse erythematous eruption with a yellowish tinge and many definite, small pinpoint-sized, atrophic pits, giving the whole eruption a reticulated appearance. The reaction to a tuberculin patch test was positive. She received six unfiltered roentgen ray treatments two years ago.

DISCUSSION

DR. L. F. X. WILHELM: Such a condition has been reported under the name reticulated atrophy by Fordyce.

DR. C. R. ANDERSON: I think the diagnosis is atrophoderma reticulatum. Such an eruption is sometimes seen associated with von Recklinghausen's neurofibromatosis.

DR. SAMUEL AYRES JR.: The interesting thing is that when the patient first consulted us, a number of years ago, atrophy was not as apparent as it is now. At first the area of eruption was studded with dry follicular plugs or scales showing Demodex folliculorum in large numbers, which may or may not have any bearing

on the condition. At present the plugging is nearly absent and there is definite reticulated atrophy.

Linear Scleroderma. Presented by DR. NELSON PAUL ANDERSON.

I. G., a white girl aged 12 years, was presented on April 8, 1941 before this society (ARCH. DERMAT. & SYPH. **45**:191 [Jan.] 1942). The eruption had a duration of about nine years and had begun with a small spot on one shoulder, with the involvement of the fingers and thumb being noted about the same time. At the time of her previous presentation she showed a sclerodermatous thickening along the ulnar side of the left thumb. The left middle finger presented a linear thickening of the skin and subcutaneous tissues with a secondary lateral contracture of the finger toward the ulnar side. The right middle finger presented atrophy of the skin over the dorsum of the last phalanx and to a less extent over the middle knuckle. On the left side of the upper part of the chest were three dime-sized to half-dollar-sized pigmented areas with whitish and atrophic centers. The left forearm seems smaller than the right. Dr. John Stokes, who saw the patient when she was previously presented, suggested the use of an insoluble bismuth preparation. She has had twelve intramuscular injections of bismuth subsalicylate, each 2 grains (0.13 Gm.), and is presented to show the results of treatment. She now shows about 75 per cent improvement in all the affected areas.

DISCUSSION

DR. BEN A. NEWMAN: This patient presents a bilateral scalenus anticus syndrome. That is, when the head and neck are turned to one side, the radial pulse on the opposite side disappears. This indicates either the presence of a cervical rib or an unusually thickened scalenus anticus muscle. In either event, when such symptoms are present the remedy is surgical. I believe that the symmetric linear scleroderma-like lesions in this case are neurogenic, probably from pressure of some of the filaments of the brachial plexus. I suggest that the patient have roentgen ray studies for cervical ribs and detailed neurologic study for possible sensory or motor disturbances.

DR. NELSON PAUL ANDERSON: When Dr. Stokes was a guest at the meeting last spring he suggested the use of a French preparation of insoluble bismuth, which can no longer be obtained. He has recently reported excellent results in the treatment of the localized type of scleroderma (morphea) from injections of bismuth (Stokes, J. H., in discussion of Thomas, C. C.: Calcinosis and Scleroderma, ARCH. DERMAT. & SYPH. **44**:125 [July] 1941).

A Case for Diagnosis (Dermatitis Medicamentosa?). Presented by DR. SAMUEL AYRES JR.

B. C., a Mexican woman aged 28, stated that the present eruption appeared simultaneously on her arms, hands and face five weeks ago. The lesions do not itch. She took "blue pills" for pain in the back for a week or so two months ago and again one month ago. The eruption is on the backs of the hands, and all aspects of the wrists and forearms. There are two isolated lesions on the soles. The lesions consist of ill defined to fairly well defined, erythematous, buff to violaceous macules, varying in size up to approximately that of a nickel. Some of the lesions appear to be slightly darker in the center. Those on the backs of the hands show slight scaling. There is also an eruption along the nasolabial areas, on the sides and top of the lower portion of the nose and on the chin, in the transverse fold beneath the lip. These lesions appear to be composed of tiny follicular papules.

DISCUSSION

DR. KENDAL FROST: Some additional information has just been received to the effect that the Kahn and Kline reactions in this case are strongly positive.

DR. JOHN ROGERS (by invitation): I saw this patient two weeks ago. She had a pustular infection about the nose, which I thought was a seborrheic con-

dition. The Wassermann reaction was positive at that time. I did not think that the eruption on the arms had anything to do with syphilis.

Dr. Maximilian Obermayer (by invitation): I agree with Dr. Ayres in that the clinical appearance of the cutaneous changes are typical of drug eruptions of the fixed type. On being questioned, the patient gave the information that she is working in a candy factory and she admitted eating pink sweet stuffs. It is not unlikely that hypersensitivity to erythrosin, a compound which is closely related to phenolphthalein and just as prone to cause the same type of cutaneous reaction, is responsible for the eruption. Dermatitis medicamentosa of the fixed type has been reported from eating pink cake frosting and pink ice cream and from the use of pink tooth paste. As erythrosin is one of the relatively few substances capable of producing both epidermal and dermal hypersensitivity, it would be worth while testing this patient with this compound.

Dr. Samuel Ayers Jr.: The patient stated that she worked with gelatin and ate quite a bit of it. We gave her patch tests with different colored gelatins.

Note.—The eruption disappeared rapidly when antisyphilitic treatment was given and has not returned.

Book Reviews

Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases. By Richard P. Strong, M.D., Consultant in Tropical Medicine to the Massachusetts General Hospital and the Boston City Hospital. In Two Volumes. Sixth edition. Fabrikoid. Price, $21 per set. Pp. 871, with 197 illustrations; 872-1747, with 201 illustrations. Philadelphia: P. Blakiston's Son & Co., 1942.

As the interest of American physicians in tropical medicine has increased greatly since the entrance of the United States into war, the appearance of this authoritative work is indeed timely. The excellence of the previous editions of Admiral Stitt's treatise on tropical diseases is universally recognized. As, however, the last edition was published in 1929, it is fortunate that this work could be brought up to date and thoroughly revised by no less an authority than Dr. Richard P. Strong. An example of the necessity for this revision is shown in the case of pinta, as the conception of this disease has been revolutionized within the past four years.

The work of Strong and several of his associates in the Harvard School of Tropical Medicine has resulted in the production of a more or less new book. While the author deplored the necessity of increasing the size of the book, he felt that a more comprehensive discussion of the entire subject was needed. A certain amount of space, however, has been saved by the use of fine print for some of the more technical or disputed subjects.

The present work appears in two volumes with a total of 1,747 pages of text. It contains 2 colored plates and an adequate number of black and white illustrations, including clinical photographs, photomicrographs, drawings, charts and maps prepared by the League of Nations (showing geographic location of diseases). An appendix of 124 pages includes an index of clinical diagnosis in which symptoms are listed alphabetically and described briefly. A list of laboratory procedures in diagnosis is given in the same manner. The appendix concludes with a full discussion of tropical hygiene. In this edition special attention is given to public health problems and to the prevalence of tropical diseases in Central and South America.

The extent to which the skin is involved in tropical diseases is shown by the fact that 380 pages, or more than one fifth of the book, is concerned with diseases of special dermatologic interest. These include leprosy, yaws, pinta, cutaneous leishmaniasis, verruca peruviana, tropical ulcer, granuloma inguinale, lymphogranuloma venereum, pellagra, fungous diseases and dermatitis due to contact with arthropods, poisonous plants, fish, lizards, etc. To this list could be added tularemia, rat bite fever and schistosome dermatitis (which have been the subjects of publications by dermatologists) and filariasis, onchocerciasis, dracontiasis, dengue, typhus and other forms of rickettsial diseases, descriptions of which are included. Even for such nondermatologic diseases as plague and African trypanosomiasis, illustrations of cutaneous lesions are given. From the foregoing, it is evident that the dermatologist will find a veritable mine of information in this book.

A few minor criticisms might be made. As the book is essentially one of reference, the reviewer feels that the bibliographies should have included all recent articles whose authors were mentioned in the text. In the excellent discussion on leprosy it was disappointing to find only the barest mention of the tuberculoid type, about which Wade and others have written extensively. As stated in the preface, the author has tried to avoid dogmatic discussion but has cited differences of opinion when these existed. The reviewer, however, wishes that the author had given his own opinion about the duality or unity of yaws and syphilis after having discussed all the pros and cons at some length.

The present volume is a monumental work which reflects the greatest credit on the author and his associates. It should prove to be most valuable to the dermatologist as a book of reference. The appearance of the two volumes, including the printing, quality of paper and convenient size, is all that could be desired.

The Principles and Practice of Beauty Culture. By Florence E. Wall, A.M., F.A.I.C. Pp. 679. New York: Keystone Publications, 1941.

It is the author's hope that this book will serve "as 'A Beauty Shop Bible,' to which they [beauty culturists] may readily turn for 'chapter and verse' on anything and everything they may ever have to do during a busy career in professional beauty culture."

Since the author has the blessings of a university, being a lecturer thereat on cosmetology, this book can be accepted as expressing an enlightened attitude toward the field of beauty culture. The book gives abundant evidence of the author's struggle to compromise the conflict between beauticians and physicians over their respective fields of service by the technic of advising the beauticians what to do and say in the disputed territory, with the added warning that cooperation with physicians—dermatologists in particular—is advisable.

In monosyllabic style the book treats of the basic sciences and of what the beauticians are pleased to call cosmetic dermatology. The latter subject includes some of the more common cutaneous diseases and gives the beauty culturists' approach in their treatment. It is here that the author beats her breast hardest in behalf of the medical profession, all the while supplying the beautician with the medical jargon that may serve as window dressing for the beauty parlor operator who wants to act the physician. Neither the author nor her students can be relied on to recognize under all circumstances the simplest cutaneous diseases and should make no pretense of possessing such knowledge. It is apparent that the utter chaos throughout the United States regarding what constitutes the practice of medicine and the many laws recognizing limited rights of cosmetologists in the practice of medicine compel the author to include under "cosmetic dermatology" the treatment of diseases of the skin.

Adequate treatment is given the orthodox practices of the beauty parlor, such as cleansing of the scalp, cutting, waving and dyeing of hair and manicuring.

The book is a good one; but it is yet another step in helping the trend of laymen in allied fields toward undertaking to practice medicine under the aegis of "university training."

INDEX TO VOLUME 45

The asterisk (*) preceding the page number indicates an original article in the Archives. Subject entries are made for all articles. Author entries are made for original articles and society transactions. Biographies, Book Reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters, B, O and S, respectively.

Abdomen: See also Gastrointestinal Tract; etc.
cutis gyrata of unusual location, 168
Abnormalities and Deformities: See under names of diseases, organs and regions, as Atrophy; Ectodermal Defect; Fingers and Toes; Hair, abnormalities; Pigmentation; Skin, abnormalities; Sweat Glands; etc.
Abramowitz, E. W.: Chronic lymphangitis (syphilis) of lower lip and chin, 221
Darier's disease, 976
Erythema migrans or necrobiosis lipoidica, 779
Folliculitis decalvans associated with lichen spinulosus, 1182
Inoculation tuberculosis of skin, 214
Neurotic excoriations with pigmentation or urticaria pigmentosa, 206
Prurigo nodularis, 1187
Abscess: See also under names of organs and regions
gonococcic, of prepuce, later resembling chancre (chancre blennorrhagique of Gougerot), 623
multiple self-inflicted subcutaneous abscesses; case report, 391
Acanthosis nigricans, 235, 988
nigricans (juvenile type), 236
Acarus Scabiei: See Scabies
Acetarsone, Therapy: See Pemphigus vegetans; Pemphigus vulgaris
Achromia: See Vitiligo
Acid, Ascorbic: See Ascorbic Acid
Pantothenic: See Vitamins, B
Acne agminata; acnitis, 984
agminata; rosacea-like tuberculid of Lewandowsky or acnitis, 406
chlorhydroxyquinoline (quinolor) ointment in treatment of acne necrotica miliaris of scalp, *964
rational application for pustular acne; sulfur lotion with sulfathiazole, *762
rosacea-like tuberculid of Lewandowsky, 1003, 1211
varioliformis, 841
varioliformis: chronic folliculitis and furunculosis, 593
vulgaris, 193
vulgaris; anetoderma of Schweninger and Buzzi, 425
vulgaris, endocrine factors in, 972
vulgaris in 4 month old baby, 809
vulgaris, liver extract in treatment in tuberculous patients, *959
vulgaris, tuberculin treatment in tuberculous patients, *956
Acnitis: See Acne agminata
Acrodermatitis atrophicans, 990
continua (Hallopeau) or pustular psoriasis; result of sulfathiazole therapy, 830
hiemalis; case for diagnosis, 1021
perstans or psoriasis pustulosa, 434
Acrodynia, pink disease treated by intramuscular vitamin B_1; report of 8 cases, 972
pink disease treated by wheat germ, 773
Acropachyderma: See Skin, diseases
Acrosclerosis: See Scleroderma
Actinodermatitis: See Light, toxicity
Actinomycosis or blastomycosis, 428
sulfanilamide in therapy of, 1173
Adams, J.: Granuloma annulare, 430
Recurrent vesicular eruption of hands associated with menses and low estrogen level in blood, 429
Adenoma; multiple sebaceous adenoma; case for diagnosis, 1205
of sebaceous glands (adenoma sebaceum) with consideration of keratotic adenoma sebaceum and true adenoma of sebaceous glands, *734
Adhesive plaster, dermatitis due to transparent adhesive tape, *1052
Adipose Tissue: See Fat; Lipoma
Adrenal Preparations, adrenal cortex extract in treatment of bromide eruption and bromide intoxication, 1173
Adrenals, hemorrhage; Waterhouse-Friderichsen syndrome, 967
Ainhum, 400, 401
associated with hyperkeratosis palmaris et plantaris, *574
Air, effect of ultraviolet irradiation of air on incidence of infections in infants' hospital, 390
Alcoholism, pellagra and porphyrinuria, 1170
Alderson, H. E.: Douglass W. Montgomery, 764
Scleroderma, 612
Alexander, L. J.: Bismuth ethyl camphorate; its use in 26 cases of early syphilis, *876
Alkyl Sulfates, dermatitis of hands in housewives, 586
Allergy: See Anaphylaxis and Allergy
Allington, H. V.: Foreign body reaction; case for diagnosis, 614
Allison, J. R.: Granuloma pyogenicum treated with sulfathiazole; report of case, *579
Aloe Therapy: See Ulcers
Alopecia: See also Hair, removal
areata, extensive, of dental origin; evidence that isolated areas of alopecia may be due to ipsilateral foci of infection, *349
areata of scalp and eyebrows and psoriasis of nails or paronychia congenita, 241
areata or scleroderma, 197
cicatrisata; pseudopelade or lupus erythematosus, 239
congenital, with atrophy of nails, 821
nutritional dermatoses in rat; effect of pantothenic acid deficiency, *917
Alpiner, S.: Hemochromatosis; report of case and comment on methods of making definite diagnosis, *1128
Aluminum and Aluminum Compounds, solution of aluminum citrate as substitute for solution of aluminum acetate N. F., *514
Amersbach, J. C.: Lupus erythematosus, acute disseminated, 411
Amolsch, A. L.: Histoplasmosis with mucocutaneous manifestations; report of case, *912
Amyloidosis cutis, 402
cutis or lichen planus ocreaformis (Lieberthal), 607
Anaphylaxis and Allergy: See also under names of various diseases, as Eczema; Rhus, poisoning; etc.

Anaphylaxis and Allergy—Continued
blood histamine in cases of allergy; effect of administration of histaminase on symptoms and histamine content of blood in patients with allergy, 770
endogenous allergy, *697
inheritance in guinea pigs of susceptibility to skin sensitization with simple chemical compounds, 587
new technic for making patch tests; use of pyrex cups, *761
reagin content of spinal fluid, 769

Anderson, C. R.: Dermatitis of face and eyelids from nail polish, 193
Erythema ab igne or lichen planus, 171
Gumma of nose resistant to antisyphilitic treatment, 182
Urticaria pigmentosa acquisita, 1213

Anderson, N. P.: Chromoblastomycosis; case for diagnosis, 1214
Dermatitis factitia; case for diagnosis, 192
Dermatomyositis with extensive subcutaneous calcification, 174
Ehlers-Danlos syndrome, 189
Granuloma annulare, 170
Linear scleroderma, 174, 1217
Localized scleroderma, 191
Lupus erythematosus or leprosy, 169
Nevoxanthoendothelioma, 189
Psoriasis recalcitrant to treatment, 1215
Reticulated atrophy; case for diagnosis, 1216

Andrews, G. C.: Keratolysis exfoliativa, 1180
Lichenoid dermatitis following arsphenamine, 212
Lupus erythematosus and lipogranulomatosis, 812
Pseudoxanthoma elasticum, 1179
Rosacea-like tuberculid of Lewandowsky, 1003
Ulcus vulvae acutum, 1179

Androgens, testosterone propionate in treatment of male postclimacteric dermatoses, *356

Anemia, sickle cell, with ulcer, 240

Anesthesia, ointment for relief of discomfort due to severe irradiation epidermitis, 392

Anetoderma: See Atrophy

Aneurysm, tertiary syphilis with obstruction of azygos and hemiazygos veins by, 201

Angioid Streaks: See Retina, blood supply

Angiokeratoma of scrotum, 625

Angioma, associated facial hemangioma and intracranial lesion (Weber-Dimitri disease), 767
cavernosum; treatment with roentgen rays at close range, 167
serpiginosum, 175
Sturge-Weber syndrome; its occurrence in 2 cases, 390

Anhidrosis: See Sweat Glands

Animals, treatment of wounds caused by wild animals, 772

Anthrax, sulfanilamide therapy in 3 cases, 588

Antipyrine and renal damage, 590

Antisepsis and Antiseptics, bacterial flora of normal skin; report on effect of various ointments and solutions, with comments on clinical significance of this study, *61

Anuria: See Urine, suppression

Anus: See also Rectum
anorectal lymphogranuloma venereum in man, 602
anorectal lymphogranuloma venereum with stricture and acute arthritis, 601
granuloma inguinale with esthiomene and anal stricture; asymptomatic neurosyphilis, 602
importance of anorectal examination in campaign against venereal diseases, 974

Aortitis, annular macular syphilid, tabetic form of dementia paralytica and syphilitic aortitis, 603

Aphthae: See Stomatitis

Apparatus: See also Instruments
evaluation of methods and mechanical devices used in treatment of peripheral vascular diseases, 584
natural color lantern slides; description of apparatus, *963

Appel, B.: Lichen sclerosus et atrophicus, 426
Mycosis fungoides, 447
Pili torti; ectodermal dysplasia, 423
Sarcoid of Boeck, 412

Argyria: See under Silver

Ariboflavinosis: See Riboflavin

Arnold, H. L., Jr.: Dermatitis due to blossom of Grevillea Banksii; newly recognized and common entity in Hawaii, *1037

Arsenic and Arsenic Compounds: See also Arsphenamines; Tryparsamide
arsenical keratoses, 610, 1016
arsenical keratoses and squamous cell epitheliomas in psoriatic patient, 1016
arsenical keratoses; Bowen's precancerous dermatosis, 407
arsenical pigmentation and keratoses superimposed on dermatitis herpetiformis, 401
arsenical reaction encountered in 5 day treatment for syphilis (nephritis, hepatitis and pericarditis), 792
cutaneous manifestation from tobacco, with reference to arsenical exfoliative dermatitis, 585
dermatitis herpetiformis; arsenical pigmentation and keratosis, 1013
herpes zoster following administration of mapharsen, 1188
intravenous drip method in intensive arsenotherapy of syphilis with reference to its application for latent syphilis and for late stages of disease, *941
late syphilis with arsenical dermatitis and infectious relapse under treatment, 1210
lichen planus developing in patient under treatment for syphilis, 190
massive arsenotherapy in early syphilis; report of 60 cases in which neoarsphenamine was given, *894
massive arsenotherapy of syphilis by continuous intravenous drip method; preliminary report on 271 patients treated with arsenoxide preparation, *885
Therapy: See Syphilis; Ulcers
trivalent arsenicals in syphilis; treatment reactions, 1170

Arsenodetoxin: See Syphilis

Arsphenamines, argyria due to silver arsphenamine, 594
lichenoid dermatitis following, 212
massive arsenotherapy in early syphilis; report of 60 cases in which neoarsphenamine was given, *894
supposed incompatibility between arsphenamine and sulfanilamide, 588
syndrome of Milian's erythema of ninth day, 967

Arteries: See also Aneurysm; Periarteritis; Thrombosis; etc.
coronary, xanthomatosis of tendons and bones associated with disease of, 197
coronary, xanthomatosis with disease of, 198

Arthritis, anorectal lymphogranuloma venereum with stricture and acute arthritis, 601
arthropathic psoriasis, 591, 594, 613
chrysiasis; case for diagnosis, 1015
etiology of Reiter's disease (conjunctival-urethral-synovial syndrome), 395
urticaria pigmentosa and psoriasis, 806

Ascorbic Acid, colloid milium; vitamin deficiency disease (?), *1148
deficiency of vitamins C and B, 450
influence of vitamin C on therapeutic activity of bismuth, antimony and arsenic group of metals, 1174
influence of vitamins on infection; influence of vitamin C [ascorbic acid] deficiency on pyogenic infection, 168
lupus erythematosus disseminatus (subacute); vitamin C deficiency? 422

Asthma: See Anaphylaxis and Allergy

Astrachan, G. D.: Lymphangioma of tongue with fibrosis; case for diagnosis, 1028
Syphilitic (early) ulceration associated with Vincent's infection and osteomyelitis of toe; lymphogranuloma venereum, 621

Atrophoderma: See Atrophy

Atrophy: See also Acrodermatitis atrophicans; Poikiloderma atrophicans vasculare; and under names of organs and regions, as Nails; Nerves; etc.
anetoderma of Jadassohn, 1181
anetoderma of Schweninger and Buzzi, 422
anetoderma of Schweninger and Buzzi; acne vulgaris, 425
atrophic and pigmented patches; convulsions and hemorrhage from stomach and intestines, 210
dermatitis atrophicans lipoides diabetica; necrobiosis lipoidica diabeticorum, 154
hemiatrophy; left side of face, left thigh and left leg, 430
pseudoatrophoderma colli, 246
reticulated; case for diagnosis, 1216
with lichen planus; case for diagnosis, 797

Autohemotherapy: See Serotherapy and Hemotherapy

Avitaminoses: See under Vitamins

Ayres, S., Jr.: Acne vulgaris; case for diagnosis, 193
Dermatitis factitia; case for diagnosis, 192
Dermatitis medicamentosa; case for diagnosis, 1217
Generalized morphea, 174
Keratosis palmaris et plantaris (congenital), 1210
Lupus erythematosus resistant to treatment, 186
Necrobiosis lipoidica diabeticorum, 1210
Psoriasis recalcitrant to treatment, 1215
Reticulated atrophy; case for diagnosis, 1216
Urticaria pigmentosa, 187

BCG: See under Tuberculosis

Bacillus: See Bacteria

Bacteria, bacterial flora of normal skin; report on effect of various ointments and solutions, with comments on clinical significance of this study, *61
Leprosy: See Leprosy

Baer, R. L.: Common fungous infections of feet and groins; negligible role of exposure in causing attacks, *670

Baldness: See Alopecia

Bancroft, I.: Tuberculosis verrucosa cutis, 177

Barker, L. P.: Sarcoid or tuberculosis cutis, 407

Barwasser, N. C.: Keratosis follicularis (Darier) or keratosis follicularis contagiosa (Brooke), 246
Urticaria pigmentosa, 238

Bazin's Disease: See Erythema induratum

Bechet, P. E.: Darier's disease, 208
Dermatitis herpetiformis; arsenical pigmentation and keratosis, 1013
Dermatitis lichenoides chronica atrophicans (Csillag), 821

Bechet, P. E.—Continued
Early history of American dermatology, *482
Extensive lupus erythematosus in child following excessive exposure to sun, 216
Lichen sclerosus et atrophicus (Hallopeau), 1001
Lupus erythematosus hypertrophicus et profundus, *33

Becker, F. T.: Erythema induratum; case for diagnosis, 632

Becker, S. W.: Acanthosis nigricans (juvenile type), 236
Adenoma of sebaceous glands (adenoma sebaceum) with consideration of keratotic adenoma sebaceum and true adenoma of sebaceous glands, *734
Case for diagnosis, 248
Cutaneous manifestations of internal malignant tumors, *1069
Granuloma annulare or porokeratosis, 803
Hemorrhagic diathesis and lichen planus, 1199
Poikiloderma vasculare atrophicans or prefungoid stage of mycosis fungoides, 800
Pseudoepithelíomatous hyperplasia, benign squamous cell tumor or multiple squamous cell epithelioma, 999
Pseudopelade or lupus erythematosus, 239
Systematized nevus; case for diagnosis, 995
Trichorrhexis nodosa; case for diagnosis, 804

Belisario, J. C.: Threshold erythema dose of roentgen rays; experimental investigation of various aspects of erythema reaction and new clinical criterion for standard threshold erythema reaction, *641
Threshold erythema dose of roentgen rays; review of literature and comment on variation of doses in use in Australia, United States and England, *519

Benedek, T.: Favus, 237

Bennett, H. W. N.: Mycosis fungoides; epithelioma, 420

Benzyl Benzoate: See Scabies

Bereston, E. S.: Dermatitis due to transparent adhesive tape, *1052

Berkovsky, M.: Xeroderma pigmentosum, 233

Bernstein, E. T.: Acrosclerosis (multiple telangiectases of face and mutilation of digits), 1195
Pseudoleukemid; lymphosarcoma id; dermatitis-herpetiformis-like eruption in patient with lymphosarcoma, 837

Besnier-Boeck's Disease: See Sarcoidosis

Biographies:
Bowen, John T., *505
Bulkley, L. Duncan, *499
Draper, William H., *496
Duhring, Louis Adolphus, *494
Fordyce, John A., *503
Foster, Frank P., *496
Fox, George Henry, *496
Gilchrist, Thomas Casper, *504
Hansen, Armauer, 388
Hardaway, William A., *498
Hyde, James N., *500
Keyes, Edward L., *503
Montgomery, Frank Hugh, *500
Morrow, Prince A., *497
Piffard, Henry Granger, *491
Pollitzer, Sigmund, *494
Pusey, William Allen, *501
Ravogli, Augustus, *499
Robinson, Andrew Rose, *498
Sherwell, Samuel, *495
Stelwagon, Henry Weightman, *492
Taylor, Robert W., *504
Van Harlingen, Arthur, *495
Weisse, Faneuil D., *496
White, James Clarke, *490
Zeisler, Joseph, *501

Bismuth and Bismuth Compounds, dermatitis medicamentosa, 985
new bismuth compound identical in chemical origin with sobisminol mass, *478
Therapy: See under names of various diseases, i. e., Lupus erythematosus; Syphilis; etc.
toxic effects with anuria from single injection of bismuth preparation; report of 2 cases, 390
Blastomycosis, 251, 1206. See also Moniliasis; etc.
and blastomycosis-like infections, 1173
chromoblastomycosis; case for diagnosis, 1214
chromoblastomycosis in Cuba, *19
chromoblastomycosis; report of sixth case from continental United States, 1172
disseminated, 768
or actinomycosis, 428
Blennorrhagia: See Keratosis blennorrhagica
Blood, cholesterol; hereditary xanthomatosis; familial incidence of xanthoma tuberosum associated with hypercholesteremia and cardiovascular involvement, with report of several cases of sudden death, *1
Diseases: See Anemia; Leukemia; etc.
fats and lipoids; serum lipids in sclerema neonatorum, 1175
histamine in cases of allergy; effect of administration of histaminase on symptoms and histamine content of blood in patients with allergy, 770
iodine, analysis in patients with cutaneous diseases and its relation to vegetative nervous system, 164
pressure, low; orthostatic hypotension in case of tertiary syphilis, 201
search for bacilli in blood in lupus vulgaris during biologic and physical therapy, 588
transfusion; duration of infectivity of Treponema pallidum in citrated blood stored under conditions obtaining in blood banks, 391
transfusion; loss of virulence of Treponema pallida in citrated blood at 5° C, 391
transfusion; viability of spirochetes of syphilis and yaws in desiccated blood serum, 967
Bloom, D.: Acrodermatitis hiemalis; case for diagnosis, 1021
Epidermolysis bullosa; supernumerary auricle, 977
Granuloma annulare, 978
Hereditary hemorrhagic telangiectasia, 1180
Hereditary xanthomatosis; familial incidence of xanthoma tuberosum associated with hypercholesteremia and cardiovascular involvement, with report of several cases of sudden death, *1
Linear papular pigmented nevus; supranumerary auricle; ptosis of eyelid, 977
Pemphigus (Senear-Usher type), 781
Poikiloderma atrophicans vasculare (Jacobi), 783
Poikiloderma vasculare atrophicans; case for diagnosis, 635
Radiodermatitis in case of neurodermatitis, 1185
Sycosis lupoides, 784
Bluefarb, S. M.: Dermatitis factitia, 993
Steatocystoma multiplex, 1202
Boardman, W. P.: Dermatitis actinica; case for diagnosis, 425
Dermatitis medicamentosa (phenolphthalein), 449
Boccanegra Lopez, E. N.: Familial bullous dyskeratosis (familial benign chronic pemphigus [Hailey and Hailey]), 1030
Boeck Sarcoid: See Sarcoidosis
Bones: See also under names of bones
Diseases: See Osteitis; Osteomyelitis; etc.
Schaumann-Besnier-Boeck disease, Boeck's sarcoid (with changes in bones), 414
xanthomatosis of tendons and bones associated with disease of coronary arteries, 197
Book Reviews:
Biologic Fundamentals of Radiation Therapy: A Textbook; F. Ellinger, translated by R. Gross, 254
Cancer of Face and Mouth: Diagnosis, Treatment, Surgical Repair; V. P. Blair, S. Moore and L. T. Byars, 1033
Diseases of Women; H. S. Crossen and R. J. Crossen, 1034
Elimination Diets and Patient's Allergies; R. H. Rowe, 640
Lepra en México; J. G. Urueña, 1035
Natural Resisistance and Clinical Medicine; D. Perla and J. Marmorston, 1035
Our Sex Life: Guide and Counsellor for Everyone; F. Kahn, 1036
Principles and Practice of Beauty Culture; F. E. Wall, 1220
Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases; R. P. Strong, 1219
Xanthoma and Other Dyslipoidoses; F. D. Weidman and others, 640
Botryomycosis Hominis: See Granuloma pyogenicum
Bowen's Disease: See Cancer, precancer
Brain: See also Nervous System
calcification; associated facial hemangioma and intracranial lesion (Weber-Dimitri disease), 767
Syphilis: See under Syphilis
Breast, horny papules under breasts; case for diagnosis, 231
Milk: See under Milk
Brim, C. J.: Job's illness—pellagra, *371
Brocq's Disease: See Alopecia cicatrisata
Bromide and Bromine, adrenal cortex extract in treatment of bromide eruption and bromide intoxication, 1173
Brooke-Morrow's Disease: See Keratosis follicularis
Bubble Gum: See Chewing Gum
Bubo, Climatic: See Lymphogranuloma Venereum
Bubonulus: See Lymphogranuloma Venereum
Bullosis: See Vesication
Burns, new treatment (sulfadiazine); preliminary report, 768
Buschke's Disease: See Scleroderma
Butterworth, T.: Solution of aluminum citrate as substitute for solution of aluminum acetate N. F., *514
Calcification: See also under Brain
chalk gout; report of 2 cases with brief summary of some previously reported studies on calcinosis, 1171
dermatomyositis with extensive subcutaneous calcification, 174
Calcinosis: See Calcification
Campbell, H. S.: Hidradenitis suppurativa, 181
Cancer: See also Epithelioma; Sarcoma; Tumors; and under names of organs and regions, as Sebaceous Glands; Tongue; etc.
condyloma acuminatum (carcinomatous changes?), 808
cutaneous manifestations of internal malignant tumors, *1069

Cancer—Continued
of skin in relation to multiple malignant growths, 586
precancer; arsenical keratoses; Bowen's precancerous dermatosis, 407
precancer; Bowen's disease, 598
precancer; Bowen's disease and keratosis, 627
Cañizares, O.: Chick embryo antigen; intravenous use in diagnosis of lymphogranuloma venereum, *953
Folliculitis of chin with enlargement resembling rhinophyma; "mentophyma," *963
Cannon, A. B.: Trichorrhexis nodosa, 1194
Capillaries: See Skin, blood supply
Carate: See Pinta
Carbuncle, Malignant: See Anthrax
Carcinoma: See Cancer
Cardiovascular Diseases: See also Arteries; etc.
hereditary xanthomatosis; familial incidence of xanthoma tuberosum associated with hypercholesteremia and cardiovascular involvement, with report of several cases of sudden death, *1
Caro, M. R.: Papulonecrotic tuberculid, 237
Carpenter, C. P.: External contact with monoethyl ether of diethylene glycol (carbitol solvent), *553
Castration, cutaneous vascular and pigmentary changes in castrate and eunuchoid men, 1170
Cataract, atopic eczema or corneal ulcers with beginning cataracts, 839
Cerebrospinal Fluid, development of general paresis following essentially normal spinal fluid findings, 968
reagin content of, 769
Chalk Gout: See Calcification
Chancre, gonococcic abscess of prepuce, later resembling chancre (chancre biennorrhagique of Gougerot), 623
Lymphogranulomatous: See Lymphogranuloma Venereum
Chargin, L.: Arsenical keratoses, 1016
Erythema with pigmentation; case for diagnosis, 635
Cheever, A. W.: Mycosis fungoides, 448
Cheilitis: See under Lips
Chemotherapy: See under Syphilis
Chewing Gum, cheilitis from sensitivity to oil of cinnamon present in bubble gum, 1173
Chicago Dermatological Society, history of, *1138
Chickenpox, herpes zoster associated with generalized varicella-like eruption, 191
Chin: See Face
Chlorhydroxyquinoline: See under Acne
Cholesterol: See also Lipoids
extracellular cholesterosis or granuloma fungoides, 814
in Blood: See Blood, cholesterol
Christian-Schüller Syndrome: See Schüller-Christian Syndrome
Chromhidrosis: See Sweat Glands
Chromoblastomycosis: See Blastomycosis
Chrysiasis: See Gold and Gold Compounds
Cicatrix, lichen planus in scar, 222
minimal scarring after electrodesiccation, *1165
Cigars: See Tobacco
Cinnamon, cheilitis from sensitivity to oil of cinnamon present in bubble gum, 1173
Cipollaro, A. C.: Erythema multiforme; lichen planus, 596
Pyoderma gangraenosum, 597
Claassen, H. L.: Unilateral nickel dermatitis of left forearm caused by zippers, *578
Clijat-Petges Disease: See Poikiloderma
Climacteric, testosterone propionate in treatment of male postclimacteric dermatoses, *356
Climate: See Seasons
Clothing; outbreak of dermatitis from new resin fabric finishes, 585
Cohen, D.: Case for diagnosis, 798
Naevus striatus symmetricus of thumbs, 253
Pemphigus vegetans; continuous treatment with acetarsone, 250
Psoriasis of palms and soles; pustular psoriasis? 1203
Urticaria papulosa perstans, 991
Cold, acrodermatitis hiemalis; case for diagnosis, 1021
adiponecrosis and cold, 975
urticaria; failure of treatment with histamine and histaminase, 782
Colitis, ulcerative, with pyoderma gangraenosum, 177
Color, natural color lantern slides; description of apparatus, *963
Combes, F. C.: Chronic discoid lichenoid dermatitis associated with giant follicular lymphadenopathy, 1023
Keratosis biennorrhagica, 1007
Papulonecrotic tuberculid (lesions on palms), 1008
Pellagra (with flexural lesions), 1004
Comedones, grouped comedos following herpes zoster, 998
Complement Fixation: See also under Syphilis fixation; serologic diagnosis of syphilis; value of complement fixation and agglutination with spirochetal antigens and relation of spirochetal antibody to Wassermann reagin, *455
Condyloma acuminatum (carcinomatous changes?), 808
Congo Red: See Amyloidosis
Congress: See Societies
Conjunctiva, erythema nodosum with involvement of, 589
Conjunctivitis, etiology of Reiter's disease (conjunctival-urethral-synovial syndrome), 395
Parinaud's; conjunctival infection of lymphogranuloma venereum as cause of Parinaud's disease, 167
Constitution, human constitution and syphilitic infection; review of literature, projected method of study and preliminary results in 36 patients, 583
Convulsions: See also Epilepsy
atrophic and pigmented patches; convulsions and hemorrhage from stomach and intestines, 210
Coomber, R. B.: Dermatitis exfoliativa (type?), 245
Cooper, Z. K.: Erythema elevatum diutinum; report of case with histologic and bacteriologic studies, *334
Cornbleet, T.: Acquired multiple pigmented nevi, 787
Case for diagnosis, 790, 798
Lymphoblastoma or parapsoriasis, 1203
Multiple sebaceous adenoma; case for diagnosis, 1205
Psoriasis of palms and soles; pustular psoriasis? 1203
Scleroderma, 796
Seborrheic eczema treated with 40 per cent sulfur paste, 789
Sickle cell anemia with ulcer, 240

Cornea, atopic eczema or corneal ulcers with beginning cataracts, 839
Inflammation: See Keratitis
molluscum contagiosum corneae, 1177
Syphilis: See Keratitis, interstitial

Cornu Cutaneum: See Horns, cutaneous

Corynebacterium Diphtheriae: See Diphtheria

Cosmetics, dermatitis due to face powder conveyor, 1172
dermatitis due to liquid lip rouge, *152
dermatitis of face and eyelids from nail polish, 193
nail lacquer dermatitis, 1171
nail polish dermatitis, 769
new emulsion type of hand lotion for dermatologic use, *153

Costello, M. J.: Argyria due to silver arsphenamine, 594
Dermatitis herpetiformis controlled with sulfathiazole, 226
Dermatomyositis, diffuse and generalized, 403
Erythema multiforme, 595
Follic ulitis capitis marginalis, 981
Herpes zoster following administration of mapharsen, 1188
Interstitial keratitis (late congenital syphilis), 409
Lupus vulgaris with complicating synovitis, 222
Multiple epithelioma superimposed on lupus erythematosus, 1189
Papulonecrotic tuberculid, 1026
Pseudoxanthoma elasticum with angioid streaks, improved by liver therapy, 1185
Pustular psoriasis, 224
Pyoderma gangraenosum, 1022
Reticulated hyperpigmentation and depigmentation of face, 219
Reticulated hyperpigmentation; case for diagnosis, 408
Rosacea-like tuberculid of Lewandowsky or acnitis, 406
Subcutaneous fat necrosis of newborn, 409

Cowpox, milkers' nodules, 1171

Cranch, A. G.: External contact with monoethyl ether of diethylene glycol (carbitol solvent), *553

Crawford, G. M.: Blastomycosis or actinomycosis, 428
Poikiloderma atrophicans vasculare (Jacobi); parapsoriasis, 431
Sarcoma of Kaposi, 429

Creams, rational prescription writing in dermatology, *1101

Crocodiles, treatment of wounds caused by wild animals, 772

Csillag's Disease: See Dermatitis lichenoides chronica atrophicans

Curth, W.: Hepatosplenomegaly, probably due to lymphogranuloma venereum, 1192

Curtis, G. H.: Discrete keratoderma (Michael), 432
Generalized sycosis vulgaris (lupoid type), 436
Persistent nonpitting edema of eyelids; elephantiasis due to lymphogranuloma venereum, 435
Psoriasis pustulosa or acrodermatitis perstans, 434
Pustular psoriasis, acrodermatitis continua (Hallopeau); result of sulfathiazole therapy, 830
Scleroderma, lymphangioma or nevus, 829
Trophoneurosis, 438
Ulerythema acneiforme, 437
Xanthoma or lupus erythematosus with xanthomatous infiltration, 440
Xanthoma tuberosum multiplex, 433

Cutis Gyrata: See Skin, abnormalities
Hyperelastica: See Skin, abnormalities

Cylindroma, tumors on lower extremities and malignant changes in ulcers of leg; cylindroma in case of ulcus cruris, 396

Cysts: See under names of organs and regions

Dandruff: See Seborrhea

Danlos-Ehlers Syndrome: See Joints; Skin, abnormalities; Tumors

Darier's Disease: See Keratosis follicularis

Darling's Disease: See Histoplasmosis

Death, sudden; hereditary xanthomatosis; familial incidence of xanthoma tuberosum associated with hypercholesteremia and cardiovascular involvement, with report of several cases of sudden death, *1

Deficiency Diseases: See Pellagra; Vitamins; etc.

Degeneration, colloid milium; vitamin deficiency disease (?), *1148

Dementia Paralytica; annular macular syphilid, tabetic form of dementia paralytica and syphilitic aortitis, 603
artificial fever therapy in juvenile neurosyphilis, *688
development of general paresis following essentially normal spinal fluid findings, 968
in patient 9 years old, 421
infantilis et juvenilis, 590

De Oreo, G.: Lichen planopilaris; lichen planus et acuminatus atrophicans (Feldman) and lichen spinulosus and folliculitis decalvans (Little), *1081

Depigmentation: See Pigmentation

Depilation: See Hair, removal

Depilatories: See Hair, removal

Dermatitis: See also Acrodermatitis; Eczema; Neurodermatitis; etc.
Acneform: See Acne
Actinica: See Light, toxicity
Arsphenamine: See Arsphenamines
Atopic: See Eczema; Neurodermatitis
Atrophicans: See Atrophy
Blastomycetic: See Blastomycosis
chairman's address, 585
chronic discoid lichenoid dermatitis associated with giant follicular lymphadenopathy, 1023
chronic, exudative, discoid and lichenoid dermatitis, 776
chronic purpuric lichenoid dermatitis of Gougerot and Blum, 399
Contact: See Dermatitis venenata
cutaneous manifestation from tobacco, with reference to arsenical exfoliative dermatitis, 585
Eczematous: See Eczema
exfoliativa; relation of hepatic damage to cutaneous disease; 9 cases of erythroderma exfoliativum generalisatum, 166
exfoliativa; Ritter's disease; report of case in which recovery occurred, 767
exfoliativa (type?), 245
exfoliative (lymphoblastoma?), 441
external contact with monoethyl ether of diethylene glycol (carbitol solvent), *553
factitia, 192, 993
factitia and epitheliomas, 608
factitia; necrotic excoriations with pigmentation or urticaria pigmentosa, 206
factitia or post-traumatic neuromyelopathy with trophic ulceration, 200
herpetiformis, 447, 801, 1000, 1196
herpetiformis; analysis of blood iodine in patients with cutaneous diseases and its relation to vegetative nervous system, 164
herpetiformis, arsenical pigmentation and keratoses superimposed on, 401
herpetiformis; arsenical pigmentation and keratosis, 1013

Dermatitis—Continued
herpetiformis controlled with sulfathiazole, 226
herpetiformis; pseudoleukemid; lymphosarcoma id; dermatitis-herpetiformis-like eruption in patient with lymphosarcoma, 837
herpetiformis; results of treatment with sulfathiazole, 405
lichenoid, following arsphenamine, 212
lichenoides chronica atrophicans (Csillag), 622, 821
Medicamentosa: See also under Drugs; and under names of drugs, as Arsenic and Arsenic Compounds; Arsphenamines; Bismuth and Bismuth Compounds; Gold and Gold Compounds; Phenolphthalein and Phenolphthalein Derivatives; Silver; Tryparsamide; etc.
medicamentosa; case for diagnosis, 1217
medicamentosa, papulonecrotic tuberculid or leukemid, 1190
Multiformis: See Dermatitis herpetiformis
new preparation (siccolam) of metallic oxides with dehydrating and antiphlogistic effects on skin, 587
nodularis necrotica, 445
Occupational: See Industrial Diseases
of eyelids due to philodendron (Scandens cardatum) plants, *1066
of hands in housewives, 586
oral administration of ragweed oil in patients hypersensitive to ragweed oil, 584
papillaris capillitii, 838
pigmented purpuric lichenoid dermatitis of Gougerot and Blum, 232
Rhus: See Rhus, poisoning
Roentgen Ray: See under Roentgen Rays
Schamberg's disease or pigmented purpuric lichenoid dermatitis, 840
Seborrheic: See Seborrhea
Solar: See under Light
toxic; case for diagnosis, 247
Venenata: See also under Gold and Gold Compounds; Nickel; Plants; Rubber; etc.
venenata, caused by tar paper storage bags; report of 2 cases, *383
venenata; contact dermatitis; case for diagnosis, 188, 198
venenata; contact dermatitis; change in sensitivity, *580
venenata; contact dermatitis from emetine hydrochloride, *550
venenata; dermatitis of face and eyelids from nail polish, 193
venenata; derris root dermatitis, 586
venenata due to blossom of Grevillea Banksii; newly recognized and common entity in Hawaii, *1037
venenata due to liquid lip rouge, *152
venenata due to transparent adhesive tape, *1052
venenata from cutting oils, 970
venenata from imitation leather, *1164
venenata; nail lacquer dermatitis, 1171
venenata; nail polish dermatitis, 769
venenata or psoriasis, 397
venenata; outbreak from new resin fabric finishes, 585
venenata; unilateral nickel dermatitis of left forearm caused by zippers, *578
Dermatologic societies, directory of, 255
Dermatology, early history of American dermatology, *482
history of Chicago Dermatological Society, *1138
Dermatomycosis: See Mycosis, cutaneous
Dermatomyositis, diffuse and generalized, 403
is there a common denominator in scleroderma, dermatomyositis, disseminated lupus erythematosus, Libman-Sacks syndrome and polyarteritis nodosa? 771
with extensive subcutaneous calcification, 174
Dermatophytosis: See Mycosis, cutaneous
Dermatorrhexis: See Joints; Skin, abnormalities; Tumors
Dermatoses: See Dermatitis; Eczema; Skin, diseases; etc.
Derris root dermatitis, 586
Detergents: See also Alkyl Sulfates; Soaps
new industrial skin cleanser, 970
wetting agents and possible danger from their use, 154
Dextrose content of skin and intradermal insulin therapy in psoriasis, 588
Diabetes, Bronze: See Hemochromatosis
Diabetes Insipidus caused by Boeck's sarcoid, 1178
Diabetes Mellitus: See also Xanthoma diabeticorum
dermatitis atrophicans lipoides diabetica; necrobiosis lipoidica diabeticorum, 154
necrobiosis lipoidica diabeticorum, 991, 1210
necrobiosis lipoidica diabeticorum in juvenile diabetic patient, 837
sensitivity to protamine zinc insulin, *694
Diaphragm, esophageal obstruction due to gummata of esophagus and diaphragm, 583
Diathermy, minimal scarring after electrodesiccation, *1165
Diathesis, Hemorrhagic, 584. See also Purpura; etc.
hemorrhagic, and lichen planus, 1199
Dickel, H. A.: Artificial fever therapy in juvenile neurosyphilis, *688
Dickman, M.: Condyloma acuminatum (carcinomatous changes?), 808
Diet and Dietetics: See also Nutrition; Vitamins; and under names of food, as Milk; etc.
value of elimination diets in treatment of infantile eczema, 769
Diethylene Glycol: See Ethylene glycol
Digestive Tract: See Gastrointestinal Tract; Stomach
Diphtheria, experimental therapy with diphtheria toxoid in 4 cases, 784
secondary infection of cutaneous lesions with Corynebacterium diphtheriae, *757
Toxoid: See Eczema; Mycosis fungoides; Pemphigus; Sarcoidosis; etc.
Directory of dermatologic societies, 255
Dopa: See Hydroxyphenylalanine
Dorne, M.: Lymphoblastoma (lymphosarcoma or leukemia), 997
Necrobiosis lipoidica diabeticorum, 991
Parapsoriasis with features of poikiloderma vasculare atrophicans, 799
Dostrovsky, A.: Treatment of cutaneous leishmaniasis (oriental sore) by grenz rays, *865
Downing, J. G.: Anetoderma of Schweninger and Buzzi; acne vulgaris, 425
Deproteinated pancreatic extract in treatment of psoriasis, *1125
Dermatitis herpetiformis, 447
Kaposi's sarcoma, 452
Pemphigus foliaceus, 451
Drugs, Eruptions: See also under names of drugs, as Arsenic and Arsenic Compounds; Arsphenamines; Bismuth and Bismuth Compounds; Gold and Gold Compounds; Phenolphthalein and Phenolphthalein Derivatives; Silver; Tryparsamide; etc.
eruptions; leprosy, sarcoidosis or fixed drug eruption, 178
inheritance in guinea pigs of susceptibility to skin sensitization with simple chemical compounds, 587

Duhring's Disease: See Dermatitis herpetiformis

Durand-Nicolas-Favre's Disease: See Lymphogranuloma Venereum

Dwork, K. G.: Cutaneous leishmaniasis (oriental sore) in United States and Canada; survey of literature and report of 4 cases, *676

Dyshidrosis: See under Sweat Glands

Dyskeratosis: See Keratosis

Dystrophy: See under Nails

Ear, epidermolysis bullosa; supernumerary auricle, 977
linear papular pigmented nevus; supernumerary auricle; ptosis of eyelid, 977
otomycosis, 202
pseudoepitheliomatous hyperplasia, benign squamous cell tumor or multiple squamous cell epithelioma, 999

Ebert, M. H.: Acroscleroses, 243
Arsenical therapy for chronic ulcer, 790
Case for diagnosis, 996
Esthiomene; case for diagnosis, 1201
Lichen planus with atrophy; case for diagnosis, 797
Melanoma, 997
Pemphigus vulgaris treated with acetarsone, 796

Ectodermal Defect, persons lacking sweat glands, 584
pili torti; ectodermal dysplasia, 423

Eczema, atopic, or corneal ulcers with beginning cataracts, 839
chronic, and moniliasis, 598
chronic (mycotic?); experimental therapy with diphtheria toxoid, 784
eczematoid dermatitis or lymphoblastoma, 799
in infants and young children, 1175
infantile: value of elimination diets in treatment, 769
nummular, 763
or lichen spinulosus, 823
poikiloderma atrophicans vasculare (Jacobi); infectious eczematoid dermatitis, 204
Seborrheic: See Seborrhea

Edema: See Elephantiasis; and under names of organs, as Eyelids; etc.

Ehlers-Danlos Syndrome: See Joints; Skin, abnormalities; Tumors

Elastorrhexis: See Skin, abnormalities

Electrocoagulation: See under specific headings, as Hair; etc.

Electrodesiccation: See Diathermy

Electrolysis: See Hair, removal

Electropyrexia: See under Syphilis

Electrosurgery: See under specific headings, as Verruca; etc.

Elephantiasis; esthiomene; case for diagnosis, 1201
lupus erythematosus with elephantiasis of lip, 1212
persistent nonpitting edema of eyelids; elephantiasis due to lymphogranuloma venereum, 435

Elimination Diet: See under Diet and Dietetics

Ellis, F. A.: Necrobiosis lipoidica and granuloma annulare; comparative study, *40

Emetine hydrochloride, contact dermatitis from, *550

Encephalitis, Vegetative: See Acrodynia

Endocarditis, verrucous; is there a common denominator in scleroderma, dermatomyositis, disseminated lupus erythematosus, Libman-Sacks syndrome and polyarteritis nodosa? 771

Endocrine Glands, Bazin's indurated erythema; probable endocrine origin, 163
endocrine factors in acne vulgaris, 972

Engeler, J. E.: Avitaminosis (pellagra and cheilitis), 205
Chronic ulcerative radiodermatitis (encouraging results following treatment with aloe vera leaf and aloe vera ointment), 199
Otomycosis, 202

Engman, M. F., Jr.: Erythema elevatum diutinum; report of case with histologic and bacteriologic studies, *334

Eosinophils, eosinophilic leukemia; case for diagnosis, 802

Epidermodysplasia Verruciformis: See Verruca

Epidermolysis bullosa, 414, 419, 778
bullosa congenita (2 cases), 615
bullosa; supernumerary auricle, 977

Epidermomycosis; Epidermophytosis: See Moniliasis; Mycosis, cutaneous; Ringworm

Epilation: See Hair, removal

Epilepsy: See also Convulsions
Sturge-Weber syndrome; its occurrence in 2 cases, 390

Epiloia, 771

Epinephrine: See Adrenal Preparations

Epithelioma adenoides cysticum (turban tumor), 431
and dermatitis factitia, 608
arsenical keratoses and squamous cell epitheliomas in psoriatic patient, 1016
atypical superficial epitheliomatosis, 395
basal cell epithelioma in lupus erythematosus, 1017
case for diagnosis, 983
multiple benign cystic epithelioma; report of 10 cases in 1 family, 1171
multiple, superimposed on lupus erythematosus, 1189
or mycosis fungoides, 420
pseudoepitheliomatous hyperplasia, benign squamous cell tumor or multiple squamous cell epithelioma, 999
roentgen ray anaplastic epithelioma on floor of nose, 227
squamous cell epithelioma of lower lip; tertiary syphilis, 178

Epstein, E.: Dermatophytosis of hands; diagnosis and prognosis, *1113
Treatment of scabies with rotenone, *950
Ulcer of scalp; case for diagnosis, 610

Epstein, N. N.: Arsenical keratoses, 610
Leprosy or sarcoid, 615
Pemphigus, 609

Epstein, S.: Lichen planus confined to oral cavity in twins; report of case, *382

Erdos-Brown, M.: Pityriasis lichenoides chronica, 999

Eruptions: See under Arsenic and Arsenic Compounds; Arsphenamines; Drugs; Skin, diseases; etc.

Erysipelas, chronic; case for diagnosis, 180
sulfanilamide in treatment; control study, 586

Erysipeloid or tinea due to Trichophyton purpureum, 1184

Erythema ab igne or lichen planus, 171
annulare centrifugum, 190, 218, 1191
elevatum diutinum; report of case with histologic and bacteriologic studies, *334
induratum (Bazin); papulonecrotic tuberculid; livedo reticularis; erythema nodosum, 636
induratum, Bazin's; probable endocrine origin, 163
induratum; case for diagnosis, 632
migrans or necrobiosis lipoidica, 779
multiforme, 595
multiforme, avitaminosis with lesions of, 591

Erythema—Continued
multiforme bullosum with involvement of mucous membranes of eyes and mouth (Stevens-Johnson disease), 771
multiforme; lichen planus, 596
nodosum; papulonecrotic tuberculid; erythema induratum (Bazin); livedo reticularis, 636
nodosum with involvement of conjunctiva, 589
scarlatiniform, with systemic reaction following inunction with mercurial ointment, 583
syndrome of Milian's erythema of ninth day, 967
threshold erythema dose of roentgen rays; experimental investigation of various aspects of erythema reaction and new clinical criterion for standard threshold erythema reaction, *641
threshold erythema dose of roentgen rays; review of literature and comment on variation of doses in use in Australia, United States and England, *519
with pigmentation; case for diagnosis, 635
Erythredema: See Acrodynia
Erythroderma Exfoliativum: See Dermatitis exfoliativa
Esophagus, obstruction due to gummata of esophagus and diaphragm, 583
Esteves, J.: Pseudoxanthoma elasticum, *283
Esthiomene: See Elephantiasis: Vulva, ulcers
Estrin, M. M.: Mycosis fungoides, 807
Estrogens: See also Hormones, sex
local treatment of chronic cutaneous ulcers with diethylstilbestrol; vascular efficacy of synthetic estrogens, 393
recurrent vesicular eruption of hands associated with menses and low estrogen level in blood, 429
therapy of tinea capitis; preliminary report, *121
Ether, external contact with monoethyl ether of diethylene glycol (carbitol solvent), *553
Ethylene glycol; external contact with monoethyl ether of diethylene glycol (carbitol solvent), *553
Eunuchoidism, cutaneous vascular and pigmentary changes in castrate and eunuchoid men, 1170
Exanthem Subitum: See Roseola infantum
Ex-Lax: See Phenolphthalein and Phenolphthalein Derivatives
Explosions, accidental tattoo, 795
Extremities, blood supply; evaluation of methods and mechanical devices used in treatment of peripheral vascular diseases, 584
Eyebrows, alopecia areata of scalp and eyebrows and psoriasis of nails or paronychia congenita, 241
Eyelashes, infection of scalp and cilia with Phthirus pubis in 19 month old baby, 390
Eyelids, dermatitis due to philodendron (Scandens cardatum) plants, *1066
dermatitis of face and eyelids from nail polish, 193
multiple pedunculated fibromas of, 838
persistent nonpitting edema of eyelids; elephantiasis due to lymphogranuloma venereum, 435
ptosis; linear papular pigmented nevus; supranumerary auricle, 977
Eyes: See also Vision; and under names of special structures and diseases of eye, as Retina; etc.
Diseases: See also Cataract; etc.
diseases; ocular manifestations of lymphogranuloma venereum, 163
Face, associated facial hemangioma and intracranial lesion (Weber-Dimitri disease), 767
folliculitis of chin with enlargement resembling rhinophyma; "mentophyma," *963
reticulated hyperpigmentation and depigmentation of, 219
sycosiform eruption in woman, 1020
symmetric hyperpigmentation of, 439
Falk, A. B.: Arsenical reaction encountered in 5 day treatment for syphilis (nephritis, hepatitis and pericarditis), 792
Lichen planus with atrophy; case for diagnosis, 797
Sporotrichosis, 788
Fanning, J.: Leprosy; case for diagnosis, 613
Fat: See also Lipoids
adiponecrosis and cold, 975
clinical evaluation of superfatted soap, *377
in Blood: See Blood, fats and lipoids
Necrosis: See also Scleroderma
necrosis, subcutaneous, of newborn, 409
tolerance tests in psoriasis, 583
Favre-Nicolas' Disease: See Lymphogranuloma Venereum
Favus, 237
Feer's Disease: See Acrodynia
Feet: See Foot
Feigenbaum, H. L.: Pemphigus vulgaris (in seborrheic areas chiefly), 805
Feldman, S.: Basal cell epithelioma in lupus erythematosus, 1017
Horny papules under breasts; case for diagnosis, 231
Felsher, I. M.: Lupus erythematosus, 239, 1198
Ferrer, I.: Pinta; mal del pinto; carate, *843
Fetus: See also Pregnancy
possible transplacental transmission of virus of lymphogranuloma venereum, 975
Fever: See Malaria; etc.
Therapeutic: See Dementia Paralytica; Syphilis
Fibroma, multiple; case for diagnosis, 220
multiple pedunculated, of eyelids, 838
Fibrosarcoma, roentgen ray fibrosarcoma; case for diagnosis, 227
Fibrosis, lymphangioma of tongue with fibrosis; case for diagnosis, 1028
Fifth Venereal Disease: See Granuloma inguinale
Fingers and Toes: See also Foot; Hand
acrosclerosis (multiple telangiectases of face and mutilation of digits), 1195
familial vulnerability of special sites towards lesions of congenital syphilis; ischemic disturbance in fingers of 2 sisters, probably due to third generation syphilis, 392
foreign body reaction; case for diagnosis, 614
naevus striatus symmetricus of thumbs, 253
Nails: See Nails
syphilitic (early) ulceration associated with Vincent's infection and osteomyelitis of toe; lymphogranuloma venereum, 621
tropical ulcer of toes, 772
Finnerud, C. W.: Dermatitis exfoliativa (type?), 245
Eczematoid dermatitis or lymphoblastoma, 799
Fisher, A. A.: Chlorhydroxyquinoline (quinolor) ointment in treatment of acne necrotica miliaris of scalp, *964
Folliculitis capitis marginalis, 981
chronic, and furunculosis; acne varioliformis, 593
decalvans associated with lichen spinulosus, 1182

Folliculitis—Continued
decalvans; lichen planopilaris; lichen planus et acuminatus atrophicans (Feldman) and lichen spinulosus and folliculitis decalvans (Little), *1081
of chin with enlargement resembling rhinophyma; "mentophyma," *963
perforating, of nose, 235

Foot, N. C.: Papular urticaria pigmentosa; report of case, *906

Foot: See also Fingers and Toes; etc.
common fungous infections of feet and groins; negligible role of exposure in causing attacks, *670
dermatophytosis of feet with dermatophytid of hands, 777

Forearm, unilateral nickel dermatitis of left forearm caused by zippers, *578

Foreign Bodies, accidental tattoo, 795
foreign body reaction; case for diagnosis, 614

Foreskin: See Penis

Formaldehyde, Therapy: See Stomatitis, gangrenous

Fourth Venereal Disease: See Lymphogranuloma Venereum

Fox, H.: Atrophic and pigmented patches; convulsions and hemorrhage from stomach and intestines, 210
Common warts cured by mental suggestion, 214

Frambesia, viability of spirochetes of syphilis and yaws in desiccated blood serum, 967

Frank, L. J.: Pemphigus; case for diagnosis, 988

Frank, S. B.: Dyskeratoid dermatosis, *129
Electrosurgical removal of plantar warts, *328

Fraser, J. F.: Granuloma annulare, 811, 1015
Nonsuppurating panniculitis (Weber-Christian), 810

Freeman, H. E.: Avitaminosis (pellagra and cheilitis), 205
Chronic ulcerative radiodermatitis (encouraging results following treatment with aloe vera leaf and aloe vera ointment), 199
Keratosis blennorrhagica, 204
Orthostatic hypotension in case of tertiary syphilis, 201
Otomycosis, 202
Poikiloderma atrophicans vasculare (Jacobi); infectious eczematoid dermatitis, 204
Post-traumatic neuromyelopathy with trophic ulceration or dermatitis factitia, 200
Tertiary syphilis; pseudoepitheliomatous hyperplasia in gumma of tongue; pulmonary tuberculosis, 203
Tertiary syphilis with obstruction of azygos and hemiazygos veins by aneurysm, 201

Frei Reaction: See Lymphogranuloma Venereum

Friderichsen-Waterhouse Syndrome: See Adrenals, hemorrhage

Frost, K.: Leprosy, sarcoidosis or fixed drug eruption, 178
Palmar lichen planus, 171

Fruchtbaum, L. M.: Acnitis, 984
Lichen planus, 808

Fungi: See also Actinomycosis; Blastomycosis; Mycosis; Ringworm; Trichophytosis; etc.
common fungous infections of feet and groins; negligible role of exposure in causing attacks, *670
lupus erythematosus complicated by fungous infection, 187

Furunculosis and chronic folliculitis; acne varioliformis, 593

Fusospirochetosis, syphilitic (early) ulceration associated with Vincent's infection and osteomyelitis of toe; lymphogranuloma venereum, 621
toxic effects with anuria from single injection of bismuth preparation; report of 2 cases, 390

Gahan, E.: Geographic distribution of lupus erythematosus, *1133
Lupus erythematosus; clinical observations in 443 cases, *685

Garb, J.: Xanthoma diabeticorum with unusual form of eruption; report of case, *723

Garfield, W. T.: Deficiency of vitamins C and B, 450
Lichen spinulosus; vitamin A deficiency, 423
Polyvitamin deficiency; lichen spinulosus, 424
Syphilis acquired at age of 4 years; gumma of palate, 451

Gastrointestinal Tract: See also Intestines; Rectum; Stomach; etc.
gastrointestinal changes in pemphigus, 395
new bismuth compound identical in chemical origin with sobisminol mass, *478

Genitals: See also under names of genitals, as Penis; Vagina; etc.
generalized pityriasis rosea, with palmar, plantar and genital lesions, 832, 1021
treatment of venereal warts with their extracts; preliminary report, 588

Geography, geographic distribution of lupus erythematosus, *1133

Ginsberg, J. E.: Dermatologic aspects of roentgen ray field distribution, *364

Glicklich, E. A.: Deproteinated pancreatic extract in treatment of psoriasis, *1125

Gold, M. A.: Blastomycosis, 1206
Case for diagnosis, 993
Lymphoblastoma, 1208

Gold and Gold Compounds, chrysiasis; case for diagnosis, 1015
lupus erythematosus complicated by dermatitis medicamentosa (gold) or avitaminosis, 442
Therapy: See under Lupus erythematosus; Syphilis; Tuberculosis; etc.

Goldberger, L. A.: Acrosclerosis (multiple telangiectases of face and mutilation of digits), 1195

Goldman, L.: Secondary infection of cutaneous lesions with Corynebacterium diphtheriae, *757
Unilateral nickel dermatitis of left forearm caused by zippers, *578

Goldman, M.: Histologic evaluation of effects of ichthammol, *933

Goldstein, M. B.: Familial lichen planus; report of 3 cases in 1 family, *1166

Goldstein's Disease: See Telangiectasia

Gonorrhea: See also Venereal Diseases
gonococcic abscess of prepuce, later resembling chancre (chancre blennorrhagique of Gougerot), 623

Gonorrhea—Continued
supposed incompatibility between arsphenamine and sulfanilamide, 588
Goodman, H.: Rational prescription writing in dermatology, *1101
Gordon Test: See under Hodgkin's Disease
Gout, Calcified: See Calcification
Grace, J. D.: Extensive alopecia areata of dental origin; evidence that isolated areas of alopecia may be due to ipsilateral foci of infection, *349
Graham, T. N.: Amyloidosis cutis, 402
Arthropathic psoriasis, 594
Papular urticaria pigmentosa; report of case, *906
Pigmentation of skin due to vitamin A deficiency; report of case and review of literature, *1156
Trichotillomania, 595
Granuloma: See also Lipogranulomatosis
annulare, 170, 217, 430, 811, 818, 978, 1015
annulare and necrobiosis lipoidica; comparative study, *40
annulare, giant syphiloid, 973
annulare or porokeratosis, 803
chronic migrating pyoderma associated with psoriasis simulating specific granuloma, 611
Fungoides: See Mycosis fungoides
Inguinale: See also Lymphogranuloma Venereum
inguinale with esthiomene and anal stricture; asymptomatic neurosyphilis, 602
Malignant: See Hodgkin's Disease
pyogenicum treated with sulfathiazole; report of case, *579
Tropicum: See Frambesia
Venereal: See Granuloma inguinale
Granulosis rubra nasi, 233
Grauer, F. H.: Juvenile tabes dorsalis with bilateral primary atrophy of optic nerve and Charcot spine, 592
Lupus vulgaris erythematodes (Leloir), 398
Greases, rational prescription writing in dermatology, *1101
Grenz Rays: See Leishmaniasis
Grevillea Banksii: See Plants
Grönblad-Strandberg Syndrome: See Pseudoxanthoma elasticum; Retina
Groin, common fungous infections of feet and groins; negligible role of exposure in causing attacks, *670
Gross, P.: Lupus erythematosus of face associated with psoriasis of legs cured with parenteral liver therapy, 231
Gumma: See Diaphragm; Esophagus; Nose; Palate; Tongue; etc.
Gums, dermatitis medicamentosa from dilantin sodium (unilateral acneform eruption and hyperplastic overgrowth of gums); Horner's syndrome, 427

Habermann's Disease: See Pityriasis lichenoides et varioliformis acuta
Hair, abnormalities; monilethrix; koilonychia, 209
abnormalities; pili torti; ectodermal dysplasia, 423
abnormalities; twisted hair (pili torti), 448
color of; nutritional dermatoses in rat; effect of pantothenic acid deficiency, *917
Diseases: See also Alopecia
diseases; trichorrhexis nodosa, 804, 1194
foreign body reaction; case for diagnosis, 614
Hair—Continued
hypertrichosis treated by electrocoagulation, 227
pili torti; case for diagnosis, 1183
Plucking: See Trichotillomania
removal; electrolysis; introduction of instrument for relatively painless treatment, *1094
Hall, A. F.: Lichen planus involving palms, 172
Hallopeau's Disease: See Acrodermatitis
Halloran, C.: Chronic erysipelas; case for diagnosis, 180
Dystrophy of nails due to mercury poisoning, 179
Hereditary hemorrhagic telangiectasia, 175
Resistant lupus erythematosus treated with liver extract, 169
Tuberculosis verrucosa cutis, 176
Hand, E. A.: Electrolysis; introduction of instrument for relatively painless treatment, *1094
Hand: See also Fingers and Toes
aid to diagnosis of latent syphilis-luetism, 161
dermatophytosis of; diagnosis and prognosis, *1113
dermatophytosis of feet with dermatophytid of hands, 777
new emulsion type of hand lotion for dermatologic use, *153
recurrent vesicular eruption of hands associated with menses and low estrogen level in blood, 429
Hand-Schüller-Christian Disease: See Schüller-Christian Syndrome
Hansen, Armauer, 388
Hansen's Disease: See Leprosy
Hanzlik, P. J.: New bismuth compound identical in chemical origin with sobisminol mass, *478
Haraszti, E.: Massive arsenotherapy of syphilis by continuous intravenous drip method; preliminary report on 271 patients treated with arsenoxide preparation, *885
Harris, J. H.: Dermatitis lichenoides chronica atrophicans (Csillag), 622
Dermatitis of eyelids due to philodendron (Scandens cardatum) plants, *1066
Epidermolysis bullosa congenita (2 cases), 615
Leprosy, lepromatous variety, 620
Hartman, M. S.: Pityriasis rosea, *580
Hecht, H.: Keratosis pilaris; keratosis pilaris rubra atrophicans, 600
Hecht, R.: Common fungous infections of feet and groins; negligible role of exposure in causing attacks, *670
Heerfordt's Syndrome: See Uveoparotid Fever
Heisel, E. B.: Amyloidosis cutis or lichen planus ocreaformis (Lieberthal), 607
Annular macular syphilid, tabetic form of dementia paralytica and syphilitic aortitis, 603
Anorectal lymphogranuloma venereum in man, 602
Anorectal lymphogranuloma venereum with stricture and acute arthritis, 601
Granuloma inguinale with esthiomene and anal stricture; asymptomatic neurosyphilis, 602
Keratosis blennorrhagica, 204
Parapsoriasis (pityriasis lichenoides chronica), 607

Heisel, E. B.—Continued
Pemphigus vegetans, 606
Poikiloderma atrophicans vasculare (Jacobi); infectious eczematoid dermatitis, 204
Post-traumatic neuromyelopathy with trophic ulceration or dermatitis factitia, 200
Sarcoid of Boeck; latent syphilis, 605
Tertiary syphilis; pseudoepitheliomatous hyperplasia in gumma of tongue; pulmonary tuberculosis, 203

Hemangioma, 1216
roentgen therapy of hemangiomata, 1176

Hemeralopia: See Night Blindness

Hemiatrophy: See under Atrophy

Hemochromatosis, report of case and comment on methods of making definite diagnosis, *1128

Hemorrhage: See Adrenals, hemorrhage; Diathesis, hemorrhagic; Purpura haemorrhagica; Telangiectasia; etc.

Hemotherapy: See Serotherapy and Hemotherapy

Herpes zoster accompanying meningitis in infancy, 1177
zoster associated with generalized varicella-like eruption, 191
zoster following administration of mapharsen, 1188
zoster, grouped comedos following, 998
zoster, studies on virus, 774
zoster, therapy of, 588

Hetreed, F. W.: Dermatitis herpetiformis; case for diagnosis, 1196

Hevia P., H.: Massive arsenotherapy in early syphilis; report of 60 cases in which neoarsphenamine was given, *894

Hidradenitis: See under Sweat Glands

Hinton Test: See Syphilis

Histaminase, Therapy: See Anaphylaxis and Allergy; Urticaria

Histamine, Therapy: See Urticaria

Histoplasmosis with mucocutaneous manifestations; report of case, *912

Hodgkin's Disease, cutaneous manifestations of lymphogranulomatosis benigna Schaumann, 396
lymphogranulomatosis maligna, 590

Hollander, L.: Dermatitis caused by tar paper storage bags; report of 2 cases, *383
Testosterone propionate in treatment of male postclimacteric dermatoses, *356

Hopkins, J. G.: Dermatitis herpetiformis, 1000
Extracellular cholesterosis or granuloma fungoides, 814

Hormones: See Androgens; Endocrine Glands; Estrogens; Insulin; etc.

Horner Syndrome; dermatitis medicamentosa from dilantin sodium (unilateral acneform eruption and hyperplastic overgrowth of gums), 427

Horns, cutaneous; horny papules under breasts; case for diagnosis, 231

Hospitals, effect of ultraviolet irradiation of air on incidence of infections in infants' hospital, 390

Humphrey, A. A.: Hemochromatosis; report of case and comment on methods of making definite diagnosis, *1128

Hydrogen Ion Concentration, influence of titration alkalinity of cleansing agents on *p*H of human skin, 394

Hydroxyphenylalanine; histochemical observations on melanin production in skin, *103

Hypercholesteremia: See Blood, cholesterol

Hyperkeratosis: See Keratosis

Hyperpigmentation: See Pigmentation

Hyperpyrexia: See under Dementia Paralytica; Syphilis; etc.

Hypertrichosis: See under Hair

Hypotension: See Blood pressure, low

Hypotrichosis: See Alopecia

Ichthammol, histologic evaluation of effects of, *933

Ichthyosis Simplex: See Xeroderma

Icterus: See Jaundice

Immunity: See Anaphylaxis and Allergy

Industrial Diseases, contact dermatitis from emetine hydrochloride, *550
dermatitis; chairman's address, 585
dermatitis from cutting oils, 970
derris root dermatitis, 586
first meeting of consulting staff of Dermatoses Investigations Section of United States Public Health Service, 1167

Industry and Occupations, hygiene; new industrial skin cleanser, 970

Infants, newborn; sclerema neonatorum; report of case with autopsy observations, *342
newborn, subcutaneous fat necrosis of, 409

Infection: See also Septicemia; Wounds
effect of ultraviolet irradiation of air on incidence in infants' hospital, 390
influence of vitamins; influence of vitamin B_1 [thiamine] deficiency on pyogenic infection, 167
influence of vitamins; influence of vitamin B_2 [riboflavin] deficiency on pyogenic infection, 167
influence of vitamins; influence of vitamin C [ascorbic acid] deficiency on pyogenic infection, 168
influence of vitamins; influence of vitamin D deficiency on pyogenic infection and résumé of investigations to date, 168

Infectious Diseases: See Diphtheria; Infection; Syphilis; etc.

Ingels, A. E.: Chronic migrating pyoderma associated with psoriasis simulating specific granuloma, 611

Inguinal Region: See Groin

Injections: See also Blood transfusion
intravenous drip method in intensive arsenotherapy of syphilis with reference to its application for latent syphilis and for late stages of disease, *941
massive arsenotherapy in early syphilis; report of 60 cases in which neoarsphenamine was given, *894
massive arsenotherapy of syphilis by continuous intravenous drip method; preliminary report on 271 patients treated with arsenoxide preparation, *885

Instruments: See also Apparatus
electrolysis; introduction of instrument for relatively painless treatment, *1094

Insulin: See also Diabetes Mellitus
dextrose content of skin and intradermal insulin therapy in psoriasis, 588
sensitivity to protamine zinc insulin, *694

Internal Secretions: See Endocrine Glands
Internships, military and naval service for medical students and interns, 157
military services of medical students, interns and residents, 156
Intestines: See also Gastrointestinal Tract; Rectum
atrophic and pigmented patches; convulsions and hemorrhage from stomach and intestines, 210
Iodine and Iodine Compounds, in Blood: See Blood, iodine
Iris, nodulate iritis (iritis Boeck?); 3 cases, 1177
Irvine, H. G.: Parapsoriasis, 630
Isaak, I.: Treatment of cutaneous disease with radon ointment and radium pads; preliminary report, *560
Ivy Poisoning: See Rhus, poisoning

Jacobi-Petges' Disease: See Poikiloderma atrophicans vasculare
Jacobson, H. P.: Lupus erythematosus; case for diagnosis, 1213
Jadassohn's Disease: See Atrophy
Jensen, A. K.: Lichen planus developing in patient under treatment for syphilis, 190
Purpura haemorrhagica, 185
Job's illness—pellagra, *371
Johnson-Stevens Disease: See Erythema multiforme
Joints, Ehlers-Danlos syndrome, 189

Kahn, D.: Cutaneous manifestations of internal malignant tumors, *1069
Systematized nevus; case for diagnosis, 995
Trichorrhexis nodosa; case for diagnosis, 804
Kahn Test: See under Syphilis
Kala-Azar: See Leishmaniasis
Kaliski, S. R.: Estrogen therapy of tinea capitis; preliminary report, *121
Kaplan, B. I.: Intravenous drip method in intensive arsenotherapy of syphilis with reference to its application for latent syphilis and for late stages of disease, *941
Kaposi's Disease: See Xeroderma pigmentosum
Sarcoma: See Sarcoma
Karp, F. L.: Electrosurgical removal of plantar warts, *328
Kaufman, S. R.: Hereditary xanthomatosis; familial incidence of xanthoma tuberosum associated with hypercholesteremia and cardiovascular involvement, with report of several cases of sudden death, *1
Keil, H.: Dermatitis due to transparent adhesive tape, *1052
Kelly, R. J.: Avitaminosis with lesions of erythema multiforme, 591
Keloid: See also Cicatrix
xanthoma diabeticorum with unusual form of eruption; report of case, *723
Keratitis, interstitial; analysis of 532 cases with reference to standardization of treatment, 971
interstitial (late congenital syphilis), 409
Keratoderma: See Keratosis
Keratolysis exfoliativa, 1180
Keratoma: See Keratosis
Keratosis, adenoma of sebaceous glands (adenoma sebaceum) with consideration of keratotic adenoma sebaceum and true adenoma of sebaceous glands, *734
ainhum associated with hyperkeratosis palmaris et plantaris, *574
"allowable" claims for vitamin A deficiency; relation of visual defects and skin lesions, 772
and arsenical pigmentation superimposed on dermatitis herpetiformis, 401
and Bowen's disease, 627
arsenical, 610, 1016
arsenical, and squamous cell epitheliomas in psoriatic patient, 1016
arsenical; Bowen's precancerous dermatosis, 407
blennorrhagica, 204, 1007
dermatitis herpetiformis; arsenical pigmentation and keratosis, 1013
discrete keratoderma (Michael), 432
dyskeratoid dermatosis, *129
familial bullous dyskeratosis (familial benign chronic pemphigus [Hailey and Hailey]), 1030
follicularis; Darier's disease, 208, 417, 628, 976
follicularis (Darier's disease) treated with vitamin A, 833
follicularis (Darier) or keratosis follicularis contagiosa (Brooke), 246
granuloma annulare or porokeratosis, 803
palmaris et plantaris (congenital), 1210
pilaris, generalized, 1214
pilaris; keratosis pilaris rubra atrophicans, 600
porokeratosis (Mibelli), 782, 982
Kidneys, antipyrine and renal damage, 590
Kile, R. L.: Clinical evaluation of superfatted soap, *377
Contact dermatitis from emetine hydrochloride, *550
Kirby-Smith, H.: Necrobiosis lipoidica and granuloma annulare; comparative study, *40
Klein, H. N.: Sarcoid; case for diagnosis, 839
Kline Test: See under Syphilis
Koilonychia: See Nails
Kolmer, J. A.: Serologic diagnosis of syphilis; value of complement fixation and agglutination with spirochetal antigens and relation of spirochetal antibody to Wassermann reagin, *455
Koransky, A. R.: Case for diagnosis, 794
Krause, M. E.: Amyloidosis cutis or lichen planus ocreaformis (Lieberthal), 607
Annular macular syphilid, tabetic form of dementia paralytica and syphilitic aortitis, 603
Anorectal lymphogranuloma venereum in man, 602
Anorectal lymphogranuloma venereum with stricture and acute arthritis, 601
Granuloma inguinale with esthiomene and anal stricture; asymptomatic neurosyphilis, 602
Parapsoriasis (pityriasis lichenoides chronica), 607
Pemphigus vegetans, 606
Sarcoid of Boeck; latent syphilis, 605

Labia Majora: See Vulva

Lafreniere, E.: Pemphigus foliaceous; sepsis with pemphigoid lesions, 419

Landauer, R. S.: Dermatologic aspects of roentgen ray field distribution, *364

Lane, C. G.: Epidermolysis bullosa, 419
Lichen planus or lupus erythematosus, 415
Sarcoid, 418

Lantern slides, natural color; description of apparatus, *963

Larynx, Kaposi's sarcoma with involvement of oropharynx and larynx, 438

Lawrence, H.: Dermatitis of face and eyelids from nail polish, 193

Leather, dermatitis from imitation leather, *1164

Legs: See also Extremities; Foot
tumors on lower extremities and malignant changes in ulcers of leg; cylindroma in case of ulcus cruris, 396
Ulcers: See Ulcers

Leifer, W.: Generalized pityriasis rosea, with palmar, plantar and genital lesions, 832, 1021

Leishmaniasis, cutaneous (oriental sore) in United States and Canada; survey of literature and report of 4 cases, *676
experimental studies on, 975
intradermal test of Montenegro in, 973
treatment of cutaneous leishmaniasis (oriental sore) by grenz rays, *865

Lentigo, generalized, 979

Leprosy, case for diagnosis, 613
lepra reaction, 973
lepromatous variety, 620
new test for differential diagnosis between syringomyelia and neural leprosy, 974
or lupus erythematosus, 169
or sarcoid, 615
regional, racial and familial relationships in United States, 970
sarcoidosis or fixed drug eruption, 178
testicular lesions in, 974
tuberculoid, course and prognosis of, 974
tuberculoid, or sarcoidosis, 824

Lerner, C.: Hypertrichosis treated by electrocoagulation, 227

Leukemia, eosinophilic; case for diagnosis, 802
lymphatic; leukemid, dermatitis medicamentosa or papulonecrotic tuberculid, 1190
lymphoblastoma (lymphosarcoma or leukemia), 997
myelogenous; specific cutaneous lesions in chronic myeloid leukemia; clinical significance, *897

Leukemid: See Leukemia, lymphatic

Leukocytes: See Leukemia

Leukoderma: See Vitiligo

Leukoplakia: See Tongue

Levine, B.: Bowen's disease, 598
Keratosis pilaris; keratosis pilaris rubra atrophicans, 600
Moniliasis and chronic eczema, 598

Lewe, I. A.: Papulonecrotic tuberculid, 242
Pseudoatrophoderma colli, 246

Lewis, G. M.: Cold urticaria; failure of treatment with histamine and histaminase, 782
Erythema annulare centrifugum, 218

Lewis, G. M.—Continued
Lichen planus confined to mouth, 979
Lichen planus in scar, 222
Lichen striatus, 207
Lupus vulgaris; tuberculous adenitis, 1187
Monilethrix; koilonychia, 209
Pemphigus foliaceus, 980
Tinea capitis (Trichophyton violaceum), 780
Tinea cruris (Trichophyton purpureum), 226

Libman-Sacks Syndrome: See Endocarditis, verrucous

Lice: See Pediculosis

Lichen nitidus, 626
nitidus or lichen scrofulosorum, 444
planopilaris; lichen planus et acuminatus atrophicans (Feldman) and lichen spinulosus and folliculitis decalvans (Little), *1081
planus, 632, 808
planus and hemorrhagic diathesis, 1199
planus confined to mouth, 979
planus confined to oral cavity in twins; report of case, *382
planus developing in patient under treatment for syphilis, 190
planus; eruption closely resembling lichen planus due to wheat germ, 769
planus; erythema multiforme, 596
planus erythematosus, 842
planus, familial; report of 3 cases in 1 family, *1166
planus in scar, 222
planus involving palms, 172
planus; lichen planopilaris; lichen planus et acuminatus atrophicans (Feldman) and lichen spinulosus and folliculitis decalvans (Little), *1081
planus ocreaformis (Lieberthal) or amyloidosis cutis, 607
planus or erythema ab igne, 171
planus or lupus erythematosus, 415
planus, palmar, 171
planus; treatment with vitamin B complex, 769
planus with atrophy; case for diagnosis, 797
sclerosus et atrophicus, 188, 426
sclerosus et atrophicus (Hallopeau), 1001
scrofulosorum, 444
scrofulosorum or lichen nitidus, 444
spinulosus associated with folliculitis decalvans, 1182
spinulosus; lichen planopilaris; lichen planus et acuminatus atrophicans (Feldman) and lichen spinulosus and folliculitis decalvans (Little), *1081
spinulosus or eczema, 823
spinulosus; polyvitamin deficiency, 424
spinulosus: vitamin A deficiency, 423
striatus, 207, 444
Urticatus: See Urticaria papulosa

Lichenification: See Neurodermatitis

Lichtenstein, M. R.: Liver extract in treatment of acne vulgaris in tuberculous patients, *959
Tuberculin treatment of acne vulgaris in tuberculous patients, *956

Light: See also Radiations; Ultraviolet Rays; etc.

Light—Continued
toxicity; dermatitis actinica; case for diagnosis, 425
toxicity; extensive lupus erythematosus in child following excessive exposure to sun, 216
toxicity; lupus erythematosus following exposure to sun in patient with healed tuberculous nodes and healed pulmonary tuberculosis, 617
toxicity; subacute disseminated lupus erythematosus; light-sensitive dermatitis, 196
Limarzi, L. R.: Specific cutaneous lesions in chronic myeloid leukemia; clinical significance, *897
Lindsay, H. C. L.: Contact dermatitis; case for diagnosis, 198
Lupus erythematosus complicated by fungous infection, 187
Lingua: See Tongue
Lipemia: See Blood, fats and lipoids
Lipids: See Blood, fats and lipoids; Lipoids
Lipogranulomatosis and lupus erythematosus, 812
Lipoidosis: See Lipoids; Schüller-Christian Syndrome; Xanthoma
Lipoids: See also Cholesterol; Fat
dermatitis atrophicans lipoides diabetica; necrobiosis lipoidica diabeticorum, 154
erythema migrans or necrobiosis lipoidica, 779
in Blood: See Blood, fats and lipoids
necrobiosis lipoidica, 224
necrobiosis lipoidica and granuloma annulare; comparative study, *40
necrobiosis lipoidica diabeticorum, 991, 1210
necrobiosis lipoidica diabeticorum in juvenile diabetic patient, 837
necrobiosis lipoidica without diabetes; case for diagnosis, 633
Lipoma, naevus verrucosa linearis associated with lipomatosis and possible neurofibromas, 425
Lipomatosis: See Lipoma
Lips, avitaminosis (pellagra and cheilitis), 205
cheilitis from sensitivity to oil of cinnamon present in bubble gum, 1173
chronic lymphangitis (syphilis) of lower lip and chin, 221
diseases; riboflavin deficiency; report of case in child with cure by specific treatment, 1174
lupus erythematosus with elephantiasis of, 1212
squamous cell epithelioma of lower lip; tertiary syphilis, 178
Lipschütz's Disease: See Vulva, ulcers
Lipstick: See Cosmetics
Livedo reticularis; papulonecrotic tuberculid, 173
reticularis; papulonecrotic tuberculid; erythema induratum (Bazin); erythema nodosum, 636
Liver, arsenical reaction encountered in 5 day treatment for syphilis (nephritis, hepatitis and pericarditis), 792
hepatosplenomegaly, probably due to lymphogranuloma venereum, 1192
relation of hepatic damage to cutaneous disease; 9 cases of erythroderma exfoliativum generalisatum, 166
Therapy: See Acne vulgaris; Lupus erythematosus; Pseudoxanthoma elasticum; etc.
Livingood, C. S.: Bacterial flora of normal skin; report on effect of various ointments and solutions, with comments on clinical significance of this study, *61
Lotion, new emulsion type of hand lotion for dermatologic use, *153
rational prescription writing in dermatology, *1101
Luetin Test: See under Syphilis
Lungs, pulmonary roentgenologic observations in granuloma fungoides, 165
Tuberculosis: See Tuberculosis, pulmonary
Luotest: See under Syphilis
Lupus: See also Tuberculosis
erythematosus, 239, 626, 1198, 1213
erythematosus, acute disseminated, 411
erythematosus and lipogranulomatosis, 812
erythematosus, basal cell epithelioma in, 1017
erythematosus, chronic disseminated, 222
erythematosus; clinical observations in 443 cases, *685
erythematosus complicated by dermatitis medicamentosa (gold) or avitaminosis, 442
erythematosus complicated by fungous infection, 187
erythematosus disseminatus (subacute); vitamin C deficiency, 422
erythematosus, extensive, in child following excessive exposure to sun, 216
erythematosus following exposure to sun in patient with healed tuberculous nodes and healed pulmonary tuberculosis, 617
erythematosus, geographic distribution of, *1133
erythematosus hypertrophicus et profundus, *33
erythematosus; is there a common denominator in scleroderma, dermatomyositis, disseminated lupus erythematosus, Libman-Sacks syndrome and polyarteritis nodosa? 771
erythematosus, multiple epithelioma superimposed on, 1189
erythematosus of face associated with psoriasis of legs cured with parenteral liver therapy, 231
erythematosus of mouth; treatment, 589
erythematosus or leprosy, 169
erythematosus or lichen planus, 415
erythematosus or pseudopelade, 239
erythematosus resistant to treatment, 186
erythematosus, resistant, treated with liver extract, 169
erythematosus, subacute disseminated; light-sensitive dermatitis, 196
erythematosus with elephantiasis of lip, 1212
erythematosus; xanthoma or lupus erythematosus with xanthomatous infiltration, 440
miliaris disseminatus faciei, 217
vulgaris, 172, 446, 627
vulgaris erythematodes (Leloir), 398, 453
vulgaris; search for bacilli in blood during biologic and physical therapy, 588
vulgaris; tuberculous adenitis, 1187
vulgaris with complicating synovitis, 222
Lymph Nodes: See also Leukemia, lymphatic; Lymphosarcoma
chronic discoid lichenoid dermatitis associated with giant follicular lymphadenopathy, 1023
Hand-Schüller-Christian disease; case in which lymphadenopathy was predominent feature, 391
lupus vulgaris; tuberculous adenitis, 1187

Lymphadenopathy: See Lymph Nodes
Lymphangioma of tongue with fibrosis; case for diagnosis, 1028
scleroderma or nevus, 829
Lymphangitis, chronic (syphilis) of lower lip and chin, 221
Lymphatic System: See Lymph Nodes
Lymphedema: See Elephantiasis
Lymphoblastoma, 794, 842, 1208
exfoliative dermatitis (lymphoblastoma?), 441
lymphosarcoma or leukemia, 997
or eczematoid dermatitis, 799
or parapsoriasis, 1203
Lymphogranuloma, Hodgkin's: See Hodgkin's Disease
Inguinale: See Granuloma inguinale; Lymphogranuloma Venereum
Schaumann's: See Sarcoidosis
Lymphogranuloma Venereum, anorectal, in man, 602
anorectal, with stricture and acute arthritis, 601
case for diagnosis, 987
chick embryo antigen; intravenous use in diagnosis of, *953
comparison of Frei antigens, *112
conjunctival infection of lymphogranuloma venereum as cause of Parinaud's disease, 167
hepatosplenomegaly, probably due to, 1192
ocular manifestations of, 163
persistent nonpitting edema of eyelids; elephantiasis due to lymphogranuloma venereum, 435
possible transplacental transmission of virus, 975
statistics of cases of induratio penis plastica, 163
syphilitic (early) ulceration associated with Vincent's infection and osteomyelitis of toe, 621
Lymphoma: See Hodgkin's Disease; Lymphosarcoma; etc.
Lymphopathia Venereum: See Lymphogranuloma Venereum
Lymphosarcoma; lymphoblastoma (lymphosarcoma or leukemia), 997
pseudoleukemid; lymphosarcoma id; dermatitis-herpetiformis-like eruption in patient with lymphosarcoma, 837

M & B 693: See Molluscum contagiosum
McCarthy, F. P.: Dermatitis medicamentosa from dilantin sodium (unilateral acneform eruption and hyperplastic overgrowth of gums); Horner's syndrome, 427
Epidermolysis bullosa, 414
McCarthy, L.: Differential diagnosis of parapsoriasis, *81
McDonald, F. M.: Dermatitis nodularis necrotica, 445
Lupus vulgaris, 446
MacKee, G. M.: Howard Morrow, 385
MacVicar, D.: Pemphigus foliaceus and avitaminosis, 831
Sarcoidosis or tuberculoid leprosy, 824
Madden, J. F.: Acne varioliformis, 841
Atopic eczema or corneal ulcers with beginning cataracts, 839
Cutis hyperelastica, 841
Dermatitis papillaris capillitii, 838
Lichen planus erythematosus, 842
Madden, J. F.—Continued
Lymphoblastoma; case for diagnosis, 842
Multiple pedunculated fibromas of eyelids, 838
Schamberg's disease, 840
Schamberg's disease or pigmented purpuric lichenoid dermatitis, 840
Xanthoma tuberosum or xanthelasma, 841
Majocchi's Disease: See Purpura annularis telangiectodes
Mal del Pinto: See Pinta
Malaria, indications for therapeutic malaria in various forms of neurosyphilis, 971
technic of induced malaria as used in South Carolina State Hospital, 971
Therapeutic: See under Syphilis
Malingering, multiple self-inflicted subcutaneous abscesses; case report, 391
Mammary Gland: See Breast
Mantoux Intradermal Test: See Tuberculosis
Mapharsen: See Arsenic and Arsenic Compounds; Syphilis
Marx, J. R.: Artificial fever therapy in juvenile neurosyphilis, *688
Mason, U. G.: Tertiary syphilis with obstruction of azygos and hemiazygos veins by aneurysm, 201
Medicine, military; military and naval service for medical students and interns, 157
military; military services of medical students, interns and residents, 156
naval; military and naval service for medical students and interns, 157
Meininger, W. M.: Epitheliomas and dermatitis factitia, 608
Melanin: See also Pigmentation
excretion of melanogen in breast milk, 164
histochemical observations on melanin production in skin, *103
melanosis of Riehl, 599
Melanoblastosis cutis linearis s. systematisata, 775
Melanoderma: See Pigmentation, cutaneous
Melanogen: See Melanin
Melanoma, 997
excretion of melanogen in breast milk, 164
of central nervous system; report of 34 cases in 19 of which diagnosis was verified by operation or necropsy, 1171
Melanosis: See Melanin
Melson, R.: Armauer Hansen, 1841-1912, 388
Meningitis accompanying herpes zoster in infancy, 1177
involvement of nervous system in Besnier-Boeck-Schaumann disease, 588
Menstruation, recurrent vesicular eruption of hands associated with menses and low estrogen level in blood, 429
transmissibility of syphilis; infectiousness of vaginal secretions and menstrual blood of syphilitic women, 767
Mentophyma, folliculitis of chin with enlargement resembling rhinophyma; "mentophyma," *963
Mercury poisoning, dystrophy of nails due to, 179
scarlatiniform erythema with systemic reaction following inunction with mercurial ointment, 583
Messina, S. J.: Deproteinated pancreatic extract in treatment of psoriasis, *1125

Metals: See Gold and Gold Compounds; Mercury; Silver; etc.

Metasyphilis: See Syphilis, congenital

Metrazol, Therapy: See Neuritis, multiple

Mibelli's Disease: See Keratosis

Michelson, H. E.: Congenital syphilis (gummas), 624
Keratosis follicularis (Darier's disease), 628
Lichen nitidus, 626
Lupus vulgaris, 627
Parapsoriasis, 630
Pyoderma or pemphigus, 631

Military Medicine: See Medicine, military

Milk, excretion of melanogen in breast milk, 164

Milkers' Nodules: See Cowpox

Miller, C. S.: Necrobiosis lipoidica diabeticorum in juvenile diabetic patient, 837

Miskjian, H. G.: Symmetric hyperpigmentation of face, 439

Mitchell, J. H.: Dermatitis herpetiformis; case for diagnosis, 1196

Molds: See Fungi

Moles: See Nevi

Molluscum contagiosum and M & B 693, 972
contagiosum corneae, 1177

Monilethrix: See Hair, abnormalities

Moniliasis and chronic eczema, 598

Montenegro Test: See Leishmaniasis

Montgomery, A. H.: Mosaic warts, 619
Mosaic warts after treatment, 1028

Montgomery, H.: Verruca plana and epithelial nevus including study of epidermodysplasia verruciformis, *259

Montgomery, R. M.: Ainhum, 400, 401
Arsenical keratoses; Bowen's precancerous dermatosis, 407
Dermatophytosis and tinea glabrosa (caused by Trichophyton purpureum), 399
Mosaic warts, 619
Mosaic warts after treatment, 1028
Psoriasis arthropathica, 591

Morphea: See Scleroderma

Morris, G. E.: Chick embryo antigen; intravenous use in diagnosis of lymphogranuloma venereum, *953
Folliculitis of chin with enlargement resembling rhinophyma; "mentophyma," *963

Morrow-Brooke's Disease: See Keratosis follicularis

Mouth: See also Gums; Stomatitis; etc.
clinical and pathologic study of oral disease, based on 2,300 consecutive cases, 1172
Kaposi's sarcoma with involvement of oropharynx and larynx, 438
lichen planus confined to mouth, 979
lichen planus confined to oral cavity in twins; report of case, *382

Mucous Membrane: See also Lips; Mouth; Nose; etc.
histoplasmosis with mucocutaneous manifestations; report of case, *912
pemphigus of, 195

Muscles, post-traumatic neuromyelopathy with trophic ulceration or dermatitis factitia, 200

Mycobacterium Leprae: See under Leprosy

Mycosis: See also Actinomycosis; Blastomycosis; Moniliasis; Ringworm; Stomatitis; Tinea; etc.
cutaneous; blastomycosis and blastomycosis-like infections, 1173
cutaneous; dermatophytosis and tinea glabrosa (caused by Trichophyton purpureum), 399
cutaneous; dermatophytosis of feet with dermatophytid of hands, 777
cutaneous; dermatophytosis of hands; diagnosis and prognosis, *1113
fungoides, 241, 420, 447, 448, 794, 807, 1200
fungoides, d'emblee form, 628
fungoides; epithelioma, 420
fungoides; experimental therapy with diphtheria toxoid, 785, 786
fungoides; granuloma fungoides or extracellular cholesterosis, 814
fungoides, prefungoid stage, or poikiloderma vasculare atrophicans, 800
fungoides; pulmonary roentgenologic observations in granuloma fungoides, 165
otomycosis, 202

Myelosis, Aleukemic: See Leukemia, myelogenous

Myoblastoma, so-called, of skin, 1209

Myocarditis, xanthomatosis of tendons and bones associated with disease of coronary arteries, 197

Myxedema, localized; case for diagnosis, 234

Myxoma, roentgen treatment of myxomatous cutaneous cysts, 392

Nail Polish: See Cosmetics

Nails: See also Paronychia
alopecia areata of scalp and eyebrows and psoriasis of nails or paronychia congenita, 241
atrophy, with congenital alopecia, 821
dystrophy due to mercury poisoning, 179
monilethrix; koilonychia, 209

Nares: See Nose

Navies: See Medicine, naval

Necrobiosis lipoidica, 224
lipoidica and granuloma annulare; comparative study, *40
lipoidica diabeticorum, 991, 1210
lipoidica diabeticorum; dermatitis atrophicans lipoides diabetica, 154
lipoidica diabeticorum in juvenile diabetic patient, 837
lipoidica or erythema migrans, 779
lipoidica without diabetes; case for diagnosis, 633

Necrosis: See under Fat; Radiations

Neoarsphenamine: See Arsphenamines; and under names of various diseases, as Syphilis; etc.

Neoplasms: See Cancer; Sarcoma; Tumors

Nephritis, arsenical reaction encountered in 5 day treatment for syphilis (nephritis, hepatitis and pericarditis), 792

Nerves: See also Nervous System
juvenile tabes dorsalis with bilateral primary atrophy of optic nerve and Charcot spine, 592
new test for differential diagnosis between syringomyelia and neural leprosy, 974
optic, damage in luetic patients, 161
post-traumatic neuromyelopathy with trophic ulceration or dermatitis factitia, 200

Nervous System: See also Brain; Nerves; etc.
analysis of blood iodine in patients with cutaneous diseases and its relation to vegetative nervous system, 164
involvement in Besnier-Boeck-Schaumann disease, 588
melanoma of central nervous system; report of 34 cases in 19 of which diagnosis was verified by operation or necropsy, 1171
Syphilis: See Syphilis

Nesbit, T.: Pemphigus or Senear-Usher syndrome, 184

Netherton, E. W.: Discrete keratoderma (Michael), 432
Generalized sycosis vulgaris (lupoid type), 436
Persistent nonpitting edema of eyelids; elephantiasis due to lymphogranuloma venereum, 435
Psoriasis pustulosa or acrodermatitis perstans, 434
Pustular psoriasis or acrodermatitis continua (Hallopeau); result of sulfathiazole therapy, 830
Scleroderma, lymphangioma or nevus, 829
Trophoneurosis, 438
Ulerythema acneiforme, 437
Xanthoma or lupus erythematosus with xanthomatous infiltration, 440
Xanthoma tuberosum multiplex, 433

Neuhauser, I.: Lupus erythematosus, 239

Neuritis, multiple; neuronitis produced by thallium acetate poisoning and cured with convulsant doses of metrazol, 1176

Neurodermatitis, radiodermatitis in case of, 1185

Neurofibroma; naevus verrucosa linearis associated with lipomatosis and possible neurofibromas, 425

Neuronitis: See Neuritis, multiple

Neurosyphilis: See Syphilis

Nevi: See also Angioma
acquired multiple pigmented nevi, 787
linear papular pigmented nevus; supranumerary auricle; ptosis of eyelid, 977
naevus sebaceus and sebaceous carcinoma, 162
naevus striatus symmetricus of thumbs, 253
naevus verrucosa linearis associated with lipomatosis and possible neurofibromas, 425
scleroderma or lymphangioma, 829
systematized; case for diagnosis, 995
Vascular: See Telangiectasia
verruca plana and epithelial nevus including study of epidermodysplasia verruciformis, *259

Nevoxanthoendothelioma, 189

Newborn Infants: See Infants, newborn

Newman, B. A.: Keratosis pilaris, generalized, 1214
Late syphilis with arsenical dermatitis and infectious relapse under treatment, 1210
Livedo reticularis; papulonecrotic tuberculids, 173
Lupus erythematosus with elephantiasis of lip, 1212
Lupus vulgaris, 172
Rosacea-like tuberculid of Lewandowsky, 1211
Subacute disseminated lupus erythematosus; light-sensitive dermatitis, 196
Xanthomatosis of tendons and bones associated with disease of coronary arteries, 197

Newman, B. A.—Continued
Xanthomatosis with disease of coronary arteries, 198

Nicholls, J.: Nutritional dermatoses in rat; effect of pantothenic acid deficiency, *917
Nutritional dermatoses in rat; signs and symptoms resulting from diet containing unheated dried egg white as source of protein, *295

Nichols, A. C.: Bacterial flora of normal skin; report on effect of various ointments and solutions, with comments on clinical significance of this study, *61

Nickel, unilateral nickel dermatitis of left forearm caused by zippers, *578

Nicolas-Favre Disease: See Lymphogranuloma Venereum

Nielsen, J. C.: Artificial fever therapy in juvenile neurosyphilis, *688

Night Blindness, "allowable" claims for vitamin A deficiency; relation of visual defects and skin lesions, 772

Niles, H. D.: Chronic purpuric lichenoid dermatitis of Gougerot and Blum, 399
Dermatitis venenata or psoriasis, 397
Sycosis vulgaris and ulerythema ophryogenes, 404

Nipples: See Breast

Nisbet, T.: Squamous cell epithelioma of lower lip; tertiary syphilis, 178

Nodes: See also Erythema nodosum; Panniculitis; etc.
fibrotic nodules of skin, *315

Noma: See Stomatitis, gangrenous

Nomenclature, first meeting of consulting staff of Dermatoses Investigations Section of United States Public Health Service, 1167

Norris, C. B.: Melanosis of Riehl, 599

Nose, granulosis rubra nasi, 233
gumma resistant to antisyphilitic treatment, 182
perforating folliculitis of, 235
roentgen ray anaplastic epithelioma on floor of nose, 227

Nutrition: See also Vitamins
nutritional dermatoses in rat; effect of pantothenic acid deficiency, *917
nutritional dermatoses in rat; signs and symptoms resulting from diet containing unheated dried egg white as source of protein, *295

Obermayer, M. E.: Acanthosis nigricans (juvenile type), 236
Case for diagnosis, 248
Histologic evaluation of effects of ichthammol, *933
Pseudopelade or lupus erythematosus, 239

Obituaries:
Ducrey, Augusto, 160
Montgomery, Douglass W., 582, 764
Morrow, Howard, 385

Occupational Diseases: See Industrial Diseases

Occupations: See Industry and Occupations

Oil, dermatitis from cutting oils, 970
Sulfonated: See Alkyl Sulfates

Ointments: See also Lotion; Rhus, poisoning; Skin, diseases
bacterial flora of normal skin; report on effect of various ointments and solutions, with comments on clinical significance of this study, *61

Ointments—Continued
chlorhydroxyquinoline (quinolor) ointment in treatment of acne necrotica miliaris of scalp, *964
Oliver, E. A.: Alopecia areata of scalp and eyebrows and psoriasis of nails or paronychia congenita, 241
Blastomycosis, 1206
Case for diagnosis, 794, 993
Dermatitis factitia, 993
Lupus erythematosus; case for diagnosis, 1198
Lymphoblastoma, 1208
Papulonecrotic tuberculid, 242
Pseudoatrophoderma colli, 246
Steatocystoma multiplex, 1202
Omens, D. V.: Sporotrichosis, 788
Onychia: See Nails
Onychodystrophy: See Nails
Onycholysis: See Nails
Onychomycosis: See Nails
Oppenheim, M.: Dermatitis atrophicans lipoides diabetica; necrobiosis lipoidica diabeticorum, 154
Naevus striatus symmetricus of thumbs, 253
Pemphigus vegetans; continuous treatment with acetarsone, 250
Urticaria papulosa perstans, 991
Oriental Sore: See Leishmaniasis
Osler's Disease: See Telangiectasia
Osteitis fibrosa cystica (Albright), 390
Osteomyelitis, syphilitic (early) ulceration associated with Vincent's infection and osteomyelitis of toe; lymphogranuloma venereum, 621
Otsuka, M.: Sporotrichosis, 788

Pachyperiostitis: See Periostitis
Palate, syphilis acquired at age of 4 years; gumma of palate, 451
Palligen: See Spirochaeta Pallida
Palmer, A. E.: Histoplasmosis with mucocutaneous manifestations; report of case, *912
Panaritium: See Paronychia
Pancreas, deproteinated pancreatic extract in treatment of psoriasis, *1125
Panniculitis, nonsuppurating (Weber-Christian), 810
Papilloma, roentgen radiation of virus (Shope); effect of x-rays on papilloma virus in vitro, 969
Paralysis, General: See Dementia Paralytica
Parapsoriasis, 630
differential diagnosis of, *81
en plaques, *89, 791
guttata (pityriasis lichenoides chronica), *83
lichenoides, *97
or lymphoblastoma, 1203
or persistent pityriasis rosea, 189
or pityriasis lichenoides chronica, 607
or poikiloderma atrophicans vasculare (Jacobi), 431, 987
varioliformis, *87
with features of poikiloderma vasculare atrophicans, 799
Pardo-Castello, V.: Chromoblastomycosis in Cuba, *19
Pinta; mal del pinto; carate, *843
Paresis: See Dementia Paralytica
Parinaud's Disease: See Conjunctivitis, Parinaud's
Paronychia congenita or psoriasis of nails and alopecia areata of scalp and eyebrows, 241
with granulation tissue reaction, 638
Patch Tests: See Anaphylaxis and Allergy
Pathomimesis: See Malingering
Paul, J. T.: Specific cutaneous lesions in chronic myeloid leukemia; clinical significance, *897
Peck, S. M.: Arsenical keratoses and squamous cell epitheliomas in psoriatic patient, 1016
Keratosis follicularis (Darier's disease) treated with vitamin A, 833
Sycosiform eruption of face in woman; case for diagnosis, 1020
Pediculosis, infection of scalp and cilia with Phthirus pubis in 19 month old baby, 390
Pellagra: See also Riboflavin
and porphyrinuria, 1170
avitaminosis (pellagra and cheilitis), 205
in Chosen (Korea); autopsy observations, 167
in Chosen (Korea); clinical and statistical observations in 1939, 166
Job's illness, *371
with flexural lesions, 1004, 1025
Pemphigus, 609, 988, 996
erythematodes (Senear-Usher), 791, 994
erythematosus; sulfathiazole rash, 426
experimental therapy with diphtheria toxoid, 785
familial bullous dyskeratosis (familial benign chronic pemphigus [Hailey and Hailey]), 1030
foliaceus, 451, 980
foliaceus and avitaminosis, 831
foliaceus; sepsis with pemphigoid lesions, 419
gastrointestinal changes in, 395
of mucous membrane, 195
or pyoderma, 631
or Senear-Usher syndrome, 184
Senear-Usher type, 781
solitarius cicatrisans, 587
vegetans, 606
vegetans; continuous treatment with acetarsone, 250
vulgaris (in seborrheic areas chiefly), 805
vulgaris treated with acetarsone, 796
Penis, gonococcic abscess of prepuce, later resembling chancre (chancre biennorrhagique of Gougerot), 623
statistics of cases of induratio penis plastica, 163
Periarteritis nodosa; is there a common denominator in scleroderma, dermatomyositis, disseminated lupus erythematosus, Libman-Sacks syndrome and polyarteritis nodosa? 771
Pericarditis, arsenical reaction encountered in 5 day treatment for syphilis (nephritis, hepatitis and pericarditis), 792
Perionychia: See Paronychia
Periostitis, acropachyderma with pachyperiostitis, 968
Perlstein, M. O.: Toxic dermatitis; case for diagnosis, 247
Perspiration: See Sweat Glands
Peterkin, G. A. G.: Nummular eczema, 763
Petges-Jacobi's Disease: See Poikiloderma atrophicans vasculare

Pfaff, R. O.: Erythema elevatum diutinum; report of case with histologic and bacteriologic studies, *334

Phalanges: See Fingers and Toes

Pharynx, aphthous pharyngitis, 391
tularemia with extensive pharyngitis, 967

Phenolphthalein and Phenolphthalein Derivatives, dermatitis medicamentosa, 449, 993

Phenylarsenkeratinate: See Syphilis

Philodendron: See Plants

Photodermatosis: See Light, toxicity

Photography, natural color lantern slides; description of apparatus, *963

Photosensitization: See Light, toxicity

Phthirus Pubis: See Pediculosis

Pigmentation: See also Hair; Hemochromatosis; Melanin; Nevi; Schamberg's Disease; Silver, toxicity; Xeroderma pigmentosum; etc.
and transplantation, 162
arsenical, and keratoses superimposed on dermatitis herpetiformis, 401
atrophic and pigmented patches; convulsions and hemorrhage from stomach and intestines, 210
cutaneous vascular and pigmentary changes in castrate and eunuchoid men, 1170
dermatitis herpetiformis; arsenical pigmentation and keratosis, 1013
distribution on arm and thorax of man, 391
neurotic excoriations with pigmentation or urticaria pigmentosa, 206
of skin due to vitamin A deficiency: report of case and review of literature, *1156
reticulated hyperpigmentation, 408
reticulated hyperpigmentation and depigmentation of face, 219
symmetric hyperpigmentation of face, 439
with erythema; case for diagnosis, 635

Pili Torti: See Hair, abnormalities

Pillsbury, D. M.: Bacterial flora of normal skin; report on effect of various ointments and solutions, with comments on clinical significance of this study, *61

Pines, C.: Granulosis rubra nasi, 233

Pink Disease: See Acrodynia

Pinta, investigation of presence of spirochete causing pinta in 500 cases, 163
mal del pinto; carate, *843

Pityriasis lichenoides chronica, 999
lichenoides chronica (parapsoriasis), 607
lichenoides chronica (parapsoriasis guttata), *83
lichenoides et varioliformis acuta, 215
rosea, *580
rosea, generalized, with palmar, plantar and genital lesions, 832, 1021
rosea, persistent, or parapsoriasis, 189
rubra pilaris, 253, 1012

Placenta, possible transplacental transmission of virus of lymphogranuloma venereum,.975

Plants, dermatitis due to blossom of Grevillea Banskii; newly recognized and common entity in Hawaii, *1037
dermatitis of eyelids due to philodendron (Scandens cardatum) plants, *1066

Poikiloderma atrophicans vasculare (Jacobi), 783
atrophicans vasculare (Jacobi); infectious eczematoid dermatitis, 204

Poikiloderma—Continued
atrophicans vasculare (Jacobi); parapsoriasis, 431, 987
poikilodermatomyositis, 1009
vasculare atrophicans, case for diagnosis, 635
vasculare atrophicans or prefungoid stage of mycosis fungoides, 800
vasculare atrophicans, parapsoriasis with features of, 799

Poison Ivy: See Rhus, poisoning

Poisons and Poisoning: See under names of poisonous substances, as Mercury; Thallium; etc.

Poliosis: See Hair, color of

Pollen: See Ragweed

Polyarteritis Nodosa: See Periarteritis nodosa

Porokeratosis: See Keratosis

Position: See Posture

Posture, orthostatic hypotension in case of tertiary syphilis, 201

Poth, D. O.: Estrogen therapy of tinea capitis; preliminary report, *121

Powders, rational prescription writing in dermatology, *1101

Prats G., F.: Massive arsenotherapy of syphilis by continuous intravenous drip method; preliminary report on 271 patients treated with arsenoxide preparation, *885

Pregnancy: See also Fetus
effect of syphilis and its treatment on incidence of toxemia of, 767
reliability of serologic tests for syphilis in, 165

Prepuce: See Penis

Prescriptions, rational prescription writing in dermatology, *1101

Prostitution, course of syphilis in prostitutes, 393

Protamine Zinc Insulin: See Insulin

Proteins, nutritional dermatoses in rat; signs and symptoms resulting from diet containing unheated dried egg white as source of protein, *295

Prunes, L.: Massive arsenotherapy in early syphilis; report of 60 cases in which neoarsphenamine was given, *894

Prurigo nodularis, 1187

Pseudoatrophoderma Colli: See Atrophy

Pseudoepithelioma: See Epithelioma

Pseudoleukemid; lymphosarcoma id; dermatitis-herpetiformis-like eruption in patient with lymphosarcoma, 837

Pseudopelade: See Alopecia cicatrisata

Pseudoxanthoma elasticum, *283, 450, 774, 1179
elasticum with angioid streaks, improved by liver therapy, 1185
elasticum with angioid streaks; syndrome of Groenblad and Strandberg, *1142

Psoriasis, alopecia areata of scalp and eyebrows and psoriasis of nails or paronychia congenita, 241
arsenical keratoses and squamous cell epitheliomas in psoriatic patient, 1016
arthropathic, 591, 594, 613
chronic migrating pyoderma associated with psoriasis simulating specific granuloma, 611
deproteinated pancreatic extract in treatment of, *1125

Psoriasis—Continued
dextrose content of skin and intradermal insulin therapy in, 588
fat tolerance tests in, 583
lupus erythematosus of face associated with psoriasis of legs cured with parenteral liver therapy, 231
of palms and soles; pustular psoriasis? 1203
or dermatitis venenata, 397
pustular, 224, 600
pustular, or acrodermatitis continua (Hallopeau); result of sulfathiazole therapy, 830
pustulosa or acrodermatitis perstans, 434
recalcitrant to treatment, 1215
treatment of, 585
urticaria pigmentosa and arthritis, 806
Psychotherapy, common warts cured by mental suggestion, 214
Ptosis: See Eyelids
Public Health: See also Venereal Diseases; etc.
first meeting of consulting staff of Dermatoses Investigations Section of United States Public Health Service, 1167
Pugh, R. E., Jr.: Threshold erythema dose of roentgen rays; review of literature and comment on variation of doses in use in Australia, United States and England, *519
Purpura annularis telangiectodes, 453
haemorrhagica, 185
Waterhouse-Friderichsen syndrome, 967
Pyoderma, chronic migrating pyoderma associated with psoriasis simulating specific granuloma, 611
climatophysiology and climatopathology of skin; relation of climate to epidemiology, etiology and bacteriology of pyodermas in Central Anatolia, 164
gangraenosum, 597, 1022
gangraenosum with ulcerative colitis, 177
or pemphigus, 631
Pyrex cups, new technic for making patch tests, *761

Quinolor: See under Acne

Races, regional, racial and familial relationships in leprosy in United States, 970
Radiations: See also Roentgen Rays; Ultraviolet Rays; etc.
treatment of post-radiation necrosis by radiation therapy, 587
Radiodermatitis: See under Roentgen Rays
Radium Pads: See Skin, diseases
Radon Ointment: See Skin, diseases
Ragweed, oral administration of ragweed oil in patients hypersensitive to ragweed oil, 584
Rattner, H.: Arsenical reaction encountered in 5 day treatment for syphilis (nephritis, hepatitis and pericarditis), 792
Grouped comedos following herpes zoster, 998
Lymphoblastoma (lymphosarcoma or leukemia), 997
Parapsoriasis with features of poikiloderma vasculare atrophicans, 799
Scleroderma, 796
Seborrheic eczema treated with 40 per cent sulfur paste, 789
Reagins: See Anaphylaxis and Allergy
Rectum, anorectal lymphogranuloma venereum in man, 602
anorectal lymphogranuloma venereum with stricture and acute arthritis, 601
Rectum—Continued
importance of anorectal examination in campaign against venereal diseases, 974
Stricture: See Lymphogranuloma Venereum
Reich, N. E.: Sclerema neonatorum; report of case with autopsy observations, *342
Rein, C. R.: Dyskeratoid dermatosis, *129
Perforating folliculitis of nose, 235
Roentgen ray anaplastic epithelioma on floor of nose, 227
Roentgen ray fibrosarcoma; case for diagnosis, 227
Reiter's Disease: See Arthritis; Conjunctivitis; Urethra, inflammation
Rendu-Osler's Disease: See Telangiectasia
Resins, outbreak of dermatitis from new resin fabric finishes, 585
Reticuloendothelial System: See also Liver
reticulosarcomatosis, 165
Retina, blood supply; pseudoxanthoma elasticum, *283, 1179
blood supply; pseudoxanthoma elasticum with angioid streaks, improved by liver therapy, 1185
blood supply; pseudoxanthoma elasticum with angioid streaks; syndrome of Groenblad and Strandberg, *1142
Rhinophyma, folliculitis of chin wth enlargement resembling rhinophyma; "mentophyma," *963
Rhus, poisoning; cutaneous and systemic reactions observed during oral poison ivy therapy, 770
poisoning; oral immunization for poison ivy dermatitis, 770
Riboflavin: See also Pellagra; Vitamins, B
deficiency; report of case in child with cure by specific treatment, 1174
influence of vitamins on infection; influence of vitamin B_2 [riboflavin] deficiency on pyogenic infection, 167
Ritter's disease; report of case in which recovery occurred, 767
Ringelman, N. P.: Eruption due to sulfanilylguanidine (sulfaguanidine), *353
Ringworm: See also Favus; Mycosis; Tinea; Trichophytosis; etc.
common fungous infections of feet and groins; negligible role of exposure in cause attacks, *670
Rio Leon, E.: Chromoblastomycosis in Cuba, *19
Riordan, T. J.: Folliculitis of chin with enlargement resembling rhinophyma; "mentophyma," *963
Pellagra, 1025
Ritter's Disease: See Dermatitis exfoliativa
Robertson, J. E.: Orthostatic hypotension in case of tertiary syphilis, 201
Robinson, H. M.: Comparison of Frei antigens, *112
Robinson, H. M., Jr.: Comparison of Frei antigens, *112
Robinson, L. B.: Dermatitis medicamentosa, papulonecrotic tuberculid or leukemid, 1190
Erythema annulare centrifugum; case for diagnosis, 1191
Robinson, S. S.: Angioma serpiginosum, 175
Lichen sclerosus et atrophicus, 188
Persistent pityriasis rosea or parapsoriasis, 189

Robinson, S. S.—Continued
Sarcoidosis cutis, 170
Subacute disseminated lupus erythematosus; light-sensitive dermatitis, 196
Xanthomatosis of tendons and bones associated with disease of coronary arteries, 197

Roentgen Rays: See also Radiations
chronic ulcerative radiodermatitis (encouraging results following treatment with aloe vera leaf and aloe vera ointment), 199
dermatologic aspects of roentgen ray field distribution, *364
ointment for relief of discomfort due to severe irradiation epidermitis, 392
radiodermatitis in case of neurodermatitis, 1185
roentgen radiation of papilloma virus (Shope); effect of x-rays on papilloma virus in vitro, 969
roentgen ray anaplastic epithelioma on floor of nose, 227
roentgen ray fibrosarcoma; case for diagnosis, 227
Therapy: See under names of organs, regions and diseases, as Angioma; Hemangioma; Leishmaniasis; Myxoma; Skin, diseases; etc.
threshold erythema dose; experimental investigation of various aspects of erythema reaction and new clinical criterion for standard threshold erythema reaction, *641
threshold erythema dose; review of literature and comment on variation of doses in use in Australia, United States and England, *519

Rogers, J. D.: Hemangioma, 1216
Scleroderma or alopecia areata, 197

Ronchese, F.: Leukoplakia of tongue, 449
Lupus vulgaris erythematodes (Leloir), 453
Purpura annularis telangiectodes, 453
Twisted hair (pili torti), 448

Rosen, I.: Generalized lentigo, 979
Porokeratosis (Mibelli), 782, 982

Roseola infantum (exanthem subitum), 162

Rosin, dermatitis due to transparent adhesive tape, *1052

Rostenberg, A.: Chrysiasis; case for diagnosis, 1015

Rotenone: See Scabies

Rothman, S.: Cutaneous manifestations of internal malignant tumors, *1069
Eosinophilic leukemia; case for diagnosis, 802
Hemorrhagic diathesis and lichen planus, 1199

Rubber, contact dermatitis; change in sensitivity, *580
dermatitis due to face powder conveyor, 1172

Ryan, M.: Pseudoxanthoma elasticum; case for diagnosis, 450

Sachs, W.: Epidermolysis bullosa; case for diagnosis, 778
Granuloma annulare; case for diagnosis, 217
Histochemical observations on melanin production in skin, *103
Lichen planopilaris; lichen planus et acuminatus atrophicans (Feldman) and lichen spinulosus and folliculitis decalvans (Little), *1081

Sacks-Libman Disease: See Endocarditis, verrucous

Sagher, F.: Treatment of cutaneous leishmaniasis (oriental sore) by grenz rays, *865

Salzer, B. H.: Histochemical observations on melanin production in skin, *103

Samitz, M. H.: Contact dermatitis; change in sensitivity, *580

de Sá Penella, L.: Pseudoxanthoma elasticum, *283

Sarcoidosis, 807, 983
Boeck's disease; its clinical groups and diagnosis, 768
cutaneous manifestations of lymphogranulomatosis benigna Schaumann, 396
cutis, 170
diabetes insipidus caused by Boeck's sarcoid, 1178
disseminated, treated with diphtheria toxoid, 1010
involvement of nervous system in Besnier-Boeck-Schaumann disease, 588
leprosy or fixed drug eruption, 178
nodulate iritis (iritis Boeck?); 3 cases, 1177
or tuberculoid leprosy, 824
sarcoid, 418, 839
sarcoid of Boeck, 412
sarcoid of Boeck; latent syphilis, 605
sarcoid or leprosy, 615
sarcoid or tuberculosis cutis, 407
Schaumann-Besnier-Boeck disease, Boeck's sarcoid (with changes in bones), 414
uveoparotid fever in case of Boeck's sarcoid, 1177

Sarcoma: See also Cancer; Tumors; and under names of tumors, as Fibrosarcoma; Lymphosarcoma; etc.
Kaposi's, 429, 452
Kaposi's, possible case of, 972
Kaposi's, with involvement of oropharynx and larynx, 438
reticulosarcomatosis, 165

Saunders, G. M.: Prevalence of syphilis in Virgin Islands of United States; results of serologic survey, *506

Saunders, T. S.: Minimal scarring after electrodesiccation, *1165
Natural color lantern slides; description of apparatus, *963
Rational application for pustular acne; sulfur lotion with sulfathiazole, *762

Sayer, A.: Papulonecrotic tuberculid; erythema induratum (Bazin); livedo reticularis; erythema nodosum, 636
Pigmented purpuric lichenoid dermatitis of Gougerot and Blum, 232

Scabies, treatment with benzyl benzoate; preliminary report, 1176
treatment with rotenone, *950

Scalp, chlorhydroxyquinoline (quinolor) ointment in treatment of acne necrotica miliaris of, *964
Diseases: See under names of diseases, as Favus; Ringworm; Seborrhea; Tinea; etc.
epithelioma adenoides cysticum (turban tumor), 431
folliculitis capitis marginalis, 981
infection of scalp and cilia with Phthirus pubis in 19 month old baby, 390
syringoma, 205
tinea capitis (Trichophyton violaceum), 780
ulcer; case for diagnosis, 610

Scandens Cardatum: See Plants

Scarlet Fever, scarlatiniform erythema with systemic reaction following inunction with mercurial ointment, 583

Scars: See Cicatrix

Schamberg's Disease, 840
or pigmented purpuric lichenoid dermatitis, 840
Schaumann's Disease: See Sarcoidosis
Scheer, M.: Darier's disease, 208
Pityriasis lichenoides et varioliformis acuta, 215
Xanthoma tuberosum multiplex, 211
Schiller, A. E.: New emulsion type of hand lotion for dermatologic use, *153
Schmidt, F. R.: Parapsoriasis en plaques, 791
So-called myoblastoma of skin, 1209
Schmidt, O. E. L.: Exfoliative dermatitis (lymphoblastoma?), 441
Kaposi's sarcoma with involvement of oropharynx and larynx, 438
Lichen nitidus or lichen scrofulosorum, 444
Lichen scrofulosorum, 444
Lichen striatus, 444
Schoch, A. G.: Bismuth ethyl camphorate; its use in 26 cases of early syphilis, *876
Schonberg, I. L.: Bowen's disease, 598
Dermatitis due to liquid lip rouge, *152
Moniliasis and chronic eczema, 598
Pustular psoriasis, 600
Schorr, H. C.: Case for diagnosis, 790
Lymphoblastoma or parapsoriasis, 1203
Multiple sebaceous adenoma; case for diagnosis, 1205
Mycosis fungoides; case for diagnosis, 241
Schüller-Christian Syndrome; Hand-Schüller-Christian disease; case in which lymphadenopathy was predominant feature, 391
Schur, A.: Pityriasis lichenoides et varioliformis acuta, 215
Schwartz, G.: Pemphigus erythematosus; sulfathiazole rash, 426
Schwartz, H. J.: Papular urticaria pigmentosa; report of case, *906
Schwartz, L.: First meeting of consulting staff of Dermatoses Investigations Section of United States Public Health Service, 1167
Schwartz, W. F.: Pemphigus of mucous membrane, 195
Scleredema: See Scleroderma
Sclerema: See Scleroderma
Sclerodactylia: See Scleroderma
Scleroderma, 612, 796
acrosclerosis, 243
acrosclerosis; multiple telangiectases of face and mutilation of digits, 1195
generalized morphea, 174
healed, 632
is there a common denominator in scleroderma, dermatomyositis, disseminated lupus erythematosus, Libman-Sacks syndrome and polyarteritis nodosa? 771
linear, 174, 1217
localized, 191
lymphangioma or nevus, 829
or alopecia areata, 197
progressive, with associated vitiligo, 162
sclerema neonatorum; report of case with autopsy observations, *342
serum lipids in sclerema neonatorum, 1175
Werner's syndrome; report of first necropsy and of findings in new case, 1170
Scrofuloderma: See Tuberculosis
Scrotum, angiokeratoma of, 625
Seasons, climatophysiology and climatopathology of skin; relation of climate to epidemiology, etiology and bacteriology of pyodermas in Central Anatolia, 164
Sebaceous Glands, adenoma (adenoma sebaceum) with consideration of keratotic adenoma sebaceum and true adenoma of sebaceous glands, *734
multiple sebaceous adenoma; case for diagnosis, 1205
naevus sebaceus and sebaceous carcinoma, 162
Seborrhea, pemphigus vulgaris (in seborrheic areas chiefly), 805
seborrheic eczema treated with 40 per cent sulfur paste, 789
Secretions, Internal: See Endocrine Glands
Senear, F. E.: Acanthosis nigricans, 235
Acquired multiple pigmented nevi, 787
Case for diagnosis, 801
Mycosis fungoides; case for diagnosis, 241, 1200
Papulonecrotic tuberculid, 237
Sickle cell anemia with ulcer, 240
Senear-Usher Syndrome: See Pemphigus
Septicemia: See also Infection
pemphigus foliaceous; sepsis with pemphigoid lesions, 419
Septum, Nasal: See under Nose
Serotherapy and Hemotherapy, autohemotherapy in cases of fixed serologic reactions with negative results, 968
Shaffer, L. W.: Histoplasmosis with mucocutaneous manifestations; report of case, *912
Sharlit, H.: Chronic, exudative, discoid and lichenoid dermatitis, 776
Dermatophytosis of feet with dermatophytid of hands, 777
Histochemical observations on melanin production in skin, *103
Lupus miliaris disseminatus faciei, 217
Paronychia with granulation tissue reaction, 638
Wetting agents and possible danger from their use, 154
Shepard, J. H.: Syringoma, 205
Short Waves: See under names of organs and regions
Shumate, C. A.: Psoriasis arthropathica, 613
Scleroderma, 612
Xanthoma; case for diagnosis, 615
Siccolam: See Dermatitis
Silver, H.: Epidermodysplasia verruciformis (Lewandowsky and Lutz), 836
Urticaria pigmentosa in syphilitic adult, 1018
Silver, toxicity; argyria due to silver arsphenamine, 594
Silvers, S. H.: Pseudoxanthoma elasticum with angioid streaks; syndrome of Groenblad and Strandberg, *1142
Sims, C. F.: Histochemical observations on melanin production in skin, *103
Sing Sing Prison Hospital, intravenous drip method in intensive arsenotherapy of syphilis with reference to its application for latent syphilis and for late stages of disease, *941
Skeer, J.: Epithelioma; case for diagnosis, 983
Sarcoidosis, 807

Skeleton: See Bones

Skin: See also Dermatology
Abnormalities: See also Atrophy; Ectodermal Defect; Pigmentation; etc.
abnormalities; cutis gyrata of unusual location, 168
abnormalities; cutis hyperelastica, 841
abnormalities; Ehlers-Danlos syndrome, 189
Atrophy: See Atrophy
bacterial flora of normal skin; report on effect of various ointments and solutions, with comments on clinical significance of this study, *61
blood supply; cutaneous vascular and pigmentary changes in castrate and eunuchoid men, 1170
Cancer: See Cancer; Epithelioma; Sarcoma; etc.
cutaneous manifestations of internal malignant tumors, *1069
dextrose content of skin and intradermal insulin therapy in psoriasis, 588
Diseases: See also Dermatitis; and under names of diseases, as Eczema; Herpes; Mycosis; etc.; and under names of plants and drugs, as Arsphenamines; Rhus; etc.
diseases; acropachyderma with pachyperiostitis, 968
diseases; analysis of blood iodine in patients with cutaneous diseases and its relation to vegetative nervous system, 164
diseases; case for diagnosis, 248
diseases; colloid milium; vitamin deficiency disease (?), *1148
diseases; dermatologic aspects of roentgen ray field distribution, *364
diseases; dyskeratoid dermatosis, *129
diseases; erythema migrans or necrobiosis lipoidica, 779
diseases; experimental therapy with diphtheria toxoid in 4 cases, 784
diseases; first meeting of consulting staff of Dermatoses Investigations Section of United States Public Health Service, 1167
diseases; histologic evaluation of effects of ichthammol, *933
diseases; irradiation in dermatology, 392
diseases; necrobiosis lipoidica, 224
diseases; necrobiosis lipoidica and granuloma annulare; comparative study, *40
diseases; necrobiosis lipoidica diabeticorum, 991, 1210
diseases; necrobiosis lipoidica diabeticorum in juvenile diabetic patient, 837
diseases; necrobiosis lipoidica without diabetes; case for diagnosis, 633
diseases; nutritional dermatoses in rat; effect of pantothenic acid deficiency, *917
diseases; nutritional dermatoses in rat; signs and symptoms resulting from diet containing unheated dried egg white as source of protein, *295
diseases; rational prescription writing in dermatology, *1101
diseases; secondary infection of cutaneous lesions with Corynebacterium diphtheriae, *757
diseases; solution of aluminum citrate as substitute for solution of aluminum acetate N. F., *514
diseases; testosterone propionate in treatment of male postclimacteric dermatoses, *356
diseases; threshold erythema dose of roentgen rays; experimental investigation of various aspects of erythema reaction and new clinical criterion for standard threshold erythema reaction, *641
diseases; threshold erythema dose of roentgen rays; review of literature and comment on variation of doses in use in Australia, United States and England, *519
diseases; treatment with radon ointment and radium pads; preliminary report, *560
diseases; tuberculin in dermatologic diagnosis with reference to purified protein derivative (P.P.D.), *544
diseases; xanthoma diabeticorum with unusual form of eruption; report of case, *723
Hemorrhage: See Purpura
histoplasmosis with mucocutaneous manifestations; report of case, *912
Inflammation: See Dermatitis
influence of titration alkalinity of cleansing agents on p_H of human skin, 394
manifestations due to sulfanilamide and its derivatives, 771
new emulsion type of hand lotion for dermatologic use, *153
Pigmentation: See under Pigmentation
so-called myoblastoma of, 1209
specific cutaneous lesions in chronic myeloid leukemia; clinical significance, *897
Syphilis: See Syphilids
transplantation and pigmentation, 162
Tuberculosis: See Tuberculids; Tuberculosis
Tumors: See under Tumors
Ulcers: See Ulcers
wetting agents and possible danger from their use, 154

Smallpox Vaccine: See Herpes zoster

Smith, E. M., Jr.: Pityriasis rubra pilaris, 253
Urticaria pigmentosa, 238

Smyth, H. F., Jr.: External contact with monoethyl ether of diethylene glycol (carbitol solvent), *553

Soaps: See also Alkyl Sulfates; Detergents
clinical evaluation of superfatted soap, *377
dermatitis of hands in housewives, 586
influence of titration alkalinity of cleansing agents on p_H of human skin, 394

Sobel, N.: Gonococcic abscess of prepuce, later resembling chancre (chancre biennorrhagique of Gougerot), 623

Sobisminol: See Syphilis

Social Hygiene, 157. See also Venereal Diseases

Societies, American Academy of Dermatology and Syphilology; 4th annual meeting, 581
American Board of Dermatology and Syphilology, 158, 581, 1169
American Dermatological Association and American Academy of Dermatology and Syphilology, 582
dermatologic, directory of, 255
foreign, directory of, 255
history of Chicago Dermatological Society, *1138
international, directory of, 255
local, directory of, 257
national, directory of, 255
sectional, directory of, 256
state, directory of, 256

Society Transactions:
Atlantic Dermatologic Conference, 412
Bronx Dermatological Society, 227, 633, 1015
Brooklyn Dermatological Society, 805, 983

Society Transactions—Continued
Chicago Dermatological Society, 235, 787, 990, 1196
Cleveland Dermatological Society, 199, 432, 598, 824
Los Angeles and San Francisco Dermatologic Societies, 169
Los Angeles Dermatological Society, 184, 1210
Manhattan Dermatologic Society, 206, 776, 976, 1179
Metropolitan Dermatological Society, 397, 591
Minnesota Dermatological Society, 624, 838
New England Dermatological Society, 447
New York Academy of Medicine, Section of Dermatology and Syphilis, 615, 832, 1022, 1190
New York Dermatological Society, 809, 1000
San Francisco Dermatological Society, 608

Sodium Bismuthate: See Bismuth and Bismuth Compounds
diphenylhydantoinate; dermatitis medicamentosa from dilantin sodium (unilateral acneform eruption and hyperplastic overgrowth of gums); Horner's syndrome, 427
Gold Thiosulfate: See Gold and Gold Compounds

Solutions, rational prescription writing in dermatology, *1101

Spencer, G. A.: Ainhum associated with hyperkeratosis palmaris et plantaris, *574

Spiegel, L.: Necrobiosis lipoidica without diabetes; case for diagnosis, 633

Spinal Fluid: See Cerebrospinal Fluid

Spine, juvenile tabes dorsalis with bilateral primary atrophy of optic nerve and Charcot spine, 592
syphilis, roentgenographic diagnosis of, 392

Spirochaeta, investigation of presence of spirochete causing pinta in 500 cases, 163

Spirochaeta Pallida: See also Syphilis
duration of infectivity of Treponema pallidum in citrated blood stored under conditions obtaining in blood banks, 391
loss of virulence of Treponema pallida in citrated blood at 5° C, 391
viability of spirochetes of syphilis and yaws in desiccated blood serum, 967

Splenomegaly, hepatosplenomegaly, probably due to lymphogranuloma venereum, 1192

Sporotrichosis, 788

Steatocystoma multiplex, 1202

Stenhouse, E. L.: Poikiloderma vasculare atrophicans or prefungoid stage of mycosis fungoides, 800
Pseudoepitheliomatous hyperplasia, benign squamous cell tumor or multiple squamous cell epithelioma, 999

Sterilization, bacterial flora of normal skin; report on effect of various ointments and solutions, with comments on clinical significance of this study, *61

Sterner, J. H.: New technic for making patch tests; use of pyrex cups, *761

Stevens, R. A.: Hereditary xanthomatosis; familial incidence of xanthoma tuberosum associated with hypercholesteremia and cardiovascular involvement, with report of several cases of sudden death, *1

Stevens-Johnson Disease: See Erythema multiforme

Stewart, C. D.: Histologic evaluation of effects of ichthammol, *933

Stilbestrol: See Estrogens

Stillians, A. W.: Acrodermatitis atrophicans, 990
Liver extract in treatment of acne vulgaris in tuberculous patients, *959
Pityriasis lichenoides chronica, 999
Pityriasis rubra pilaris, 253
Tuberculin treatment of acne vulgaris in tuberculous patients, *956
Urticaria pigmentosa, 238

Stomach: See also Gastrointestinal Tract
atrophic and pigmented patches; convulsions and hemorrhage from stomach and intestines, 210
syphilis of, 585

Stomatitis, acute, treatment of, 1174
gangrenous; successful treatment of noma with solution of formaldehyde; report of 6 additional cases, 767

Strandberg-Grönblad Syndrome: See Pseudoxanthoma elasticum; Retina

Strawberry Nevi: See Angioma

Streptococci: See Scarlet Fever

Stroud, G. M.: Lupus erythematosus complicated by dermatitis medicamentosa (gold) or avitaminosis, 442
Reinfection in congenital syphilis; case for diagnosis, 827

Sturge-Weber Syndrome: See Angioma; Epilepsy

Sudoriparous Glands: See under Sweat Glands

Sulfadiazine: See Burns

Sulfaguanidine: See Sulfanilamide and Sulfanilamide Derivatives

Sulfanilamide and Sulfanilamide Derivatives, eruption due to sulfanilylguanidine (sulfaguanidine), *353
pemphigus erythematosus; sulfathiazole rash, 426
skin manifestations due to sulfanilamide and its derivatives, 771
supposed incompatibility between arsphenamine and sulfanilamide, 588
Therapy: See Actinomycosis; Anthrax; Erysipelas; etc.

Sulfanilylguanidine: See Sulfanilamide and Sulfanilamide Derivatives

Sulfapyridine: See Sulfanilamide and Sulfanilamide Derivatives

Sulfathiazole: See Acne; Acrodermatitis; Dermatitis herpetiformis; Granuloma pyogenicum; Sulfanilamide and Sulfanilamide Derivatives; etc.

Sulfur, Therapy: See Acne; Seborrhea; etc.

Sullivan, M.: Nutritional dermatoses in rat; effect of pantothenic acid deficiency, *917
Nutritional dermatoses in rat; signs and symptoms resulting from diet containing unheated dried egg white as source of protein, *295

Sulzberger, M. B.: Common fungous infections of feet and groins; negligible role of exposure in causing attacks, *670

Sunburn: See Light, toxicity

Sunlight: See under Light

Suprarenal Preparations: See Adrenal Preparations

Suprarenals: See Adrenals

Swartz, J. H.: Darier's disease (keratosis follicularis), 417
Mycosis fungoides, 420

Swartz, J. H.—Continued
Schaumann-Besnier-Boeck disease, Boeck's sarcoid (with changes in bones), 414

Sweat Glands, abnormalities; persons lacking sweat glands, 584
chromhidrosis rosea, 166
hidradenitis suppurativa, 181
syringoma, 205

Sweitzer, S. E.: Angiokeratoma of scrotum, 625
Bowen's disease and keratosis, 627
Fibrotic nodules of skin, *315
Healed scleroderma, 632
Keratosis follicularis (Darier's disease), 628
Lichen planus; case for diagnosis, 632
Lupus erythematosus, 626
Mycosis fungoides, d'emblee form, 628
Parapsoriasis, 630

Swift-Selter-Feer's Disease: See Acrodynia

Sycosis lupoides, 784
sycosiform eruption of face in woman, 1020
vulgaris and ulerythema ophryogenes, 404
vulgaris, generalized (lupoid type), 436

Synovial Membrane, lupus vulgaris with complicating synovitis, 222

Syphilids, annular macular, tabetic form of dementia paralytica and syphilitic aortitis, 603
annular papular, 452
tinea circinata suggesting tertiary syphilid, 616

Syphilis: See also Chancre; Spirochaeta Pallida; Syphilids; Venereal Diseases; and under names of organs, regions and diseases, as Aortitis; Spine; Stomach; etc.
acquired at age of 4 years; gumma of palate, 451
aid to diagnosis of latent syphilis-luetism, 161
arsenical reaction encountered in 5 day treatment for syphilis (nephritis, hepatitis and pericarditis), 792
artificial fever therapy in juvenile neurosyphilis, *688
as Canadian problem, 769
autohemotherapy in cases of fixed serologic reactions with negative results, 968
biologic false positive serologic reactions in tests for, 1170
bismuth ethyl camphorate; its use in 26 cases of early syphilis, *876
chronic lymphangitis (syphilis) of lower lip and chin, 221
congenital (gummas), 624
congenital, in only 1 of twins, 1176
congenital; interstitial keratitis (late congenital syphilis), 409
congenital, reinfection in, 827
conservation of serum by drying on paper (according to method of Krivonosova) for purpose of serodiagnosis, 774
course in prostitutes, 393
duration of infectivity of Treponema pallidum in citrated blood stored under conditions obtaining in blood banks, 391
early; long term results in treatment, 1172
early, short term intensive arsenotherapy of, 968
effect of syphilis and its treatment on incidence of toxemia of pregnancy, 767
familial vulnerability of special sites towards lesions of congenital syphilis; ischemic disturbance in fingers of 2 sisters, probably due to third generation syphilis, 392

Syphilis—Continued
fate of Treponema pallidum inoculated subcutaneously into immune rabbits, 969
five-day treatment, 969
generalized pityriasis rosea, with palmar, plantar and genital lesions, 832
granuloma inguinale with esthiomene and anal stricture; asymptomatic neurosyphilis, 602
herpes zoster following administration of mapharsen, 1188
human constitution and syphilitic infection; review of literature, projected method of study and preliminary results in 36 patients, 583
indications for therapeutic malaria in various forms of neurosyphilis, 971
influence of vitamin C on therapeutic activity of bismuth, antimony and arsenic group of metals, 1174
intravenous drip method in intensive arsenotherapy of syphilis with reference to its application for latent syphilis and for late stages of disease, *941
late, with arsenical dermatitis and infectious relapse under treatment, 1210
latent; sarcoid of Boeck, 605
lichen planus developing in patient under treatment for syphilis, 190
massive arsenotherapy by continuous intravenous drip method; preliminary report on 271 patients treated with arsenoxide preparation, *885
massive arsenotherapy in early syphilis; report of 60 cases in which neoarsphenamine was given, *894
new bismuth compound identical in chemical origin with sobisminol mass, *478
optic nerve damage in luetic patients, 161
phenylarsenkeratinate (arsenodetoxin) in treatment of human syphilis, 394
prevalence in Virgin Islands of United States; results of serologic survey, *506
reliability of serologic tests for syphilis in pregnancy, 165
serologic diagnosis; value of complement fixation and agglutination with spirochetal antigens and relation of spirochetal antibody to Wassermann reagin, *455
sobisminol mass in treatment, 161
supposed incompatibility between arsphenamine and sulfanilamide, 588
syphilitic (early) ulceration associated with Vincent's infection and osteomyelitis of toe; lymphogranuloma venereum, 621
technic of induced malaria as used in South Carolina State Hospital, 971
tertiary; case for diagnosis, 1027
tertiary, orthostatic hypotension in case of, 201
tertiary; pseudoepitheliomatous hyperplasia in gumma of tongue; pulmonary tuberculosis, 203
tertiary; squamous cell epithelioma of lower lip, 178
tertiary, with obstruction of azygos and hemiazygos veins by aneurysm, 201
therapeutic methods in neurosyphilis, 161
transmissibility; infectiousness of vaginal secretions and menstrual blood of syphilitic women, 767
trivalent arsenicals; some recent advances, comparisons and evaluations, 583
trivalent arsenicals; treatment reactions, 1170
urticaria pigmentosa in syphilitic adult, 1018

Syphilis—Continued
use of bismuth compounds in, 1173
viability of spirochetes of syphilis and yaws in desiccated blood serum, 967
Syringoma, 205
Syringomyelia, new test for differential diagnosis between syringomyelia and neural leprosy, 974

Tabes Dorsalis, annular macular syphilid, tabetic form of dementia paralytica and syphilitic aortitis, 603
artificial fever therapy in juvenile neurosyphilis, *688
juvenile, with bilateral primary atrophy of optic nerve and Charcot spine, 592
Tar paper, dermatitis caused by tar paper storage bags; report of 2 cases, *383
Tattooing, accidental, 795
Tauber, E. B.: Secondary infection of cutaneous lesions with Corynebacterium diphtheriae, *757
Teeth, extensive alopecia areata of dental origin; evidence that isolated areas of alopecia may be due to ipsilateral foci of infection, *349
Telangiectasia: See also Angioma; Purpura annularis telangiectodes
acrosclerosis (multiple telangiectases of face and mutilation of digits), 1195
hereditary hemorrhagic, 175, 1180
macularis eruptiva perstans; telangiectatic and relatively pigmentless variety of urticaria pigmentosa of adults, 774
Templeton, H. J.: Foreign body reaction; case for diagnosis, 614
Tendons, xanthomatosis of tendons and bones associated with disease of coronary arteries, 197
Testes; testicular lesions in leprosy, 974
Testosterone Propionate: See Androgens
Thallium, neuronitis produced by thallium acetate poisoning and cured with convulsant doses of metrazol, 1176
Thiamine: See also Vitamins, B
influence of vitamins on infection; influence of vitamin B_1 [thiamine] deficiency on pyogenic infection, 167
pink disease treated by intramuscular vitamin B_1; report of 8 cases, 972
Thomas, C. C.: Tuberculin in dermatologic diagnosis with reference to purified protein derivative (P.P.D.), *544
Thrombocytopenia: See Purpura haemorrhagica
Thrombosis, xanthomatosis of tendons and bones associated with disease of coronary arteries, 197
Thrush: See Moniliasis
Thumbs: See Fingers and Toes
Thurmon, F. M.: Dementia paralytica in patient 9 years old, 421
Epithelioma adenoides cysticum (turban tumor), 431
Hemiatrophy; left side of face, left thigh and left leg, 430
Tinea: See also Ringworm; Trichophytosis
capitis, estrogen therapy; preliminary report, *121
capitis (Trichophyton violaceum), 780
circinata suggesting tertiary syphilid, 616
cruris (Trichophyton purpureum), 226
Tinea—Continued
Decalvans: See Alopecia areata
due to Trichophyton purpureum or erysipeloid, 1184
Favosa: See Favus
glabrosa and dermatophytosis (caused by Trichophyton purpureum), 399
Tobacco, cutaneous manifestation from tobacco, with reference to arsenical exfoliative dermatitis, 585
Toe-Nails: See Nails
Toes: See Fingers and Toes
Tolmach, J. A.: Pigmentation of skin due to vitamin A deficiency; report of case and review of literature, *1156
Tolman, M. M.: Anetoderma of Schweninger and Buzzi, 422
Lupus erythematosus disseminatus (subacute); vitamin C deficiency, 422
Naevus verrucosa linearis associated with lipomatosis and possible neurofibromas, 425
Xanthoma tuberosum multiplex, 431
Tongue, carcinoma, erroneous diagnosis of, 393
leukoplakia of, 449
lymphangioma with fibrosis; case for diagnosis, 1028
tertiary syphilis; pseudoepitheliomatous hyperplasia in gumma of tongue; pulmonary tuberculosis, 203
Toxemia: See Pregnancy
Toxicodermas: See Dermatitis, venenata; Drugs, eruptions
Traub, E. F.: Acne vulgaris in 4 month old baby, 809
Congenital alopecia with atrophy of nails, 821
Lichen spinulosus or eczema, 823
Lupus erythematosus following exposure to sun in patient with healed tuberculous nodes and healed pulmonary tuberculosis, 617
Pityriasis rubra pilaris, 1012
Tinea circinata suggesting tertiary syphilid, 616
Xanthoma tuberosum multiplex, 820
Treponema Pallidum: See Spirochaeta Pallida
Trespalacios, F.: Chromoblastomycosis in Cuba, *19
Trichophytid: See Trichophytosis
Trichophytin: See Trichophytosis
Trichophyton purpureum, dermatophytosis and tinea glabrosa caused by, 399
purpureum; tinea cruris, 226
purpureum, tinea due to, or erysipeloid, 1184
violaceum; tinea capitis, 780
Trichophytosis, clinical and experimental studies of fungous infections in Osaka and surroundings, 589
Trichorrhexis Nodosa: See Hair, diseases
Trichotillomania, 595
Trophoneurosis, 438
Tropical Medicine: See under names of tropical diseases, as Leishmaniasis; Ulcers, tropical; etc.
Tryparsamide, optic nerve damage in luetic patients, 161
rare ocular reactions to, 968
Tubercle Bacilli: See Tuberculosis
Tuberculids: See also under Tuberculosis
in children, 166

Tuberculids—Continued
papulonecrotic, 237, 242, 1026
papulonecrotic, dermatitis medicamentosa or leukemid, 1190
papulonecrotic; erythema induratum (Bazin); livedo reticularis; erythema nodosum, 636
papulonecrotic (lesions on palms), 1008
papulonecrotic; livedo reticularis, 173
rosacea-like, of Lewandowsky, 1003, 1211
rosacea-like, of Lewandowsky or acnitis, 406
statistics on cases of tuberculosis of skin observed from 1929 to 1939, 773

Tuberculin: See also under Tuberculosis
in dermatologic diagnosis with reference to purified protein derivative (P. P. D.), *544
Therapy: See Acne vulgaris

Tuberculosis: See also Tuberculids; and under names of organs and regions, as Lymph Nodes; etc.
course and prognosis of tuberculoid leprosy, 974
cutis or sarcoid, 407
inoculation tuberculosis of skin, 214
liver extract in treatment of acne vulgaris in tuberculous patients, *959
lupus erythematosus following exposure to sun in patient with healed tuberculous nodes and healed pulmonary tuberculosis, 617
primary inoculation tuberculosis of skin; its occurrence in 4 year old child, 767
pulmonary; tertiary syphilis; pseudoepithelioma tous hyperplasia in gumma of tongue, 203
sarcoidosis or tuberculoid leprosy, 824
tuberculin in dermatologic diagnosis with reference to purified protein derivative (P. P. D.), *544
tuberculin treatment of acne vulgaris in tuberculous patients, *956
verrucosa cutis, 176, 177

Tularemia with extensive pharyngitis, 967

Tulipan, L.: Tertiary syphilis; case for diagnosis, 1027

Tumors: See also Adenoma; Angioma; Cancer; Cylindroma; Epithelioma; Fibroma; Lipoma; Lymphangioma; Lymphoblastoma; Melanoma; Myxoma; Neurofibroma; Papilloma; Sarcoma; Syringoma; etc.; and under names of organs and regions, as Ear; Eyelids; Face; Legs; Scalp; Scrotum; Sebaceous Glands; Tongue; etc.
cutaneous manifestations of internal malignant tumors, *1069
Ehlers-Danlos syndrome, 189
heterologous transplantation of mammalian tumors; transfer of human tumors to alien species, 586
pseudoepitheliomatous hyperplasia, benign squamous cell tumor or multiple squamous cell epithelioma, 999

Turnacliff, D. D.: Parapsoriasis, 630

Twins, congenital syphilis in only 1 of, 1176
lichen planus confined to oral cavity; report of case, *382

Ulcers: See also under names of organs and regions, as Cornea; Fingers and Toes; Scalp; Vulva; etc.
chronic, arsenical therapy for, 790
chronic ulcerative radiodermatitis (encouraging results following treatment with aloe vera leaf and aloe vera ointment), 199

Ulcers—Continued
local treatment of chronic cutaneous ulcers with diethylstilbestrol; vascular efficacy of synthetic estrogens, 393
of leg, 393
post-traumatic neuromyelopathy with trophic ulceration or dermatitis factitia, 200
sickle cell anemia with ulcer, 240
syphilitic (early) ulceration associated with Vincent's infection and osteomyelitis of toe; lymphogranuloma venereum, 621
tropical, of toes, 772
tumors on lower extremities and malignant changes in ulcers of leg; cylindroma in case of ulcus cruris, 396

Ulerythema acneiforme, 437
ophryogenes and sycosis vulgaris, 404

Ultrashort Waves: See under names of organs, regions and diseases

Ultraviolet Rays: See also Light
effect of ultraviolet irradiation of air on incidence of infections in infants' hospital, 390
Therapy: See under names of organs, regions and diseases, as Herpes zoster; etc.

United States Public Health Service: See Public Health

Urbach, E.: Dermatitis atrophicans lipoides diabetica; necrobiosis lipoidica diabeticorum, 154
Endogenous allergy, *697

Urethra, inflammation: etiology of Reiter's disease (conjunctival-urethral-synovial syndrome), 395

Urine, pellagra and porphyrinuria, 1170
suppression; toxic effects with anuria from single injection of bismuth preparation; report of 2 cases, 390

Urticaria, case for diagnosis, 801
cold urticaria; failure of treatment with histamine and histaminase, 782
papulosa perstans, 991
pigmentosa, 187, 238
pigmentosa acquisita, 1213
pigmentosa in syphilitic adult, 1018
pigmentosa or necrotic excoriations with pigmentation, 206
pigmentosa, papular; report of case, *906
pigmentosa, psoriasis and arthritis, 806
pigmentosa; telangiectasis macularis eruptiva perstans; telangiectactic and relatively pigmentless variety of urticaria pigmentosa of adults, 774

Usher-Senear Syndrome: See Pemphigus

Uveoparotid Fever in case of Boeck's sarcoid, 1177

Vagina, transmissibility of syphilis; infectiousness of vaginal secretions and menstrual blood of syphilitic women, 767

Van Dyck, L. S.: Arsenical pigmentation and keratoses superimposed on dermatitis herpetiformis, 401
Chronic folliculitis and furunculosis; acne varioliformis, 593
Dermatitis herpetiformis; results of treatment with sulfathiazole, 405

Van Winkle, W., Jr.: New bismuth compound identical in chemical origin with sobisminol mass, *478

Varas, L. I.: Massive arsenotherapy of syphilis by continuous intravenous drip method: preliminary report on 271 patients treated with arsenoxide preparation, *885

Varicella: See Chickenpox
Veins: See also Cardiovascular System; Thrombosis; etc.
tertiary syphilis with obstruction of azygos and hemiazygos veins by aneurysm, 201
Venereal Diseases: See also Chancre; Gonorrhea; Syphilis
importance of anorectal examination in campaign against, 974
present status of prophylaxis in Republic of Argentina, 773
Verity, L. E.: Hemochromatosis; report of case and comment on methods of making definite diagnosis, *1128
Verruca, common warts cured by mental suggestion, 214
electrosurgical removal of plantar warts, *328
epidermodysplasia verruciformis (Lewandowsky and Lutz), 836
mosaic warts, 619
mosaic warts after treatment, 1028
plana and epithelial nevus including study of epidermodysplasia verruciformis, *259
treatment of venereal warts with their extracts; preliminary report, 588
Tuberculous: See under Tuberculosis
Vesication, recurrent vesicular eruption of hands associated with menses and low estrogen level in blood, 429
Vincent's Organisms: See Fusospirochetosis
Virgin Islands, prevalence of syphilis in Virgin Islands of United States; results of serologic survey, *506
Viruses: See under Herpes; Lymphogranuloma Venereum; Papilloma; etc.
Vision, rare ocular reactions to tryparasamide, 968
Vitamins: See also Ascorbic Acid; Riboflavin; Thiamine
A; "allowable" claims for deficiency; relation of visual defects and skin lesions, 772
A deficiency; lichen spinulosus, 423
A; keratosis follicularis (Darier's disease) treated with vitamin A, 833
A; nummular eczema, 763
A; pigmentation of skin due to vitamin A deficiency; report of case and review of literature, *1156
avitaminosis (pellagra and cheilitis), 205
avitaminosis with lesions of erythema multiforme, 591
B: See also Pellagra; Riboflavin; Thiamine
B; deficiency of vitamins C and B, 450
B; eruption closely resembling lichen planus due to wheat germ, 769
B; nutritional dermatoses in rat; effect of pantothenic acid deficiency, *917
B; pink disease treated by wheat germ, 773
B; treatment of lichen planus with vitamin B complex, 769
C: See Ascorbic Acid
influence on infection: influence of vitamin D deficiency on pyogenic infection and résumé of investigations to date, 168
lupus erythematosus complicated by dermatitis medicamentosa (gold) or avitaminosis, 442
nutritional dermatoses in rat; signs and symptoms resulting from diet containing unheated dried egg white as source of protein, *295
pemphigus foliaceus and avitaminosis, 831
polyvitamin deficiency; lichen spinulosus, 424
Vitiligo, leukoderma with inflammatory border, 218
progressive scleroderma with associated vitiligo, 162
Vogel, H. R.: Testosterone propionate in treatment of male postclimacteric dermatoses, *356
Vulva, esthiomene; case for diagnosis, 1201
ulcers; granuloma inguinale with esthiomene and anal stricture; asymptomatic neurosyphilis, 602
ulcus vulvae acutum, 1179

Waisman, M.: Verruca plana and epithelial nevus including study of epidermodysplasia verruciformis, *259
Walzer, A.: Acanthosis nigricans, 988
Dermatitis medicamentosa (bismuth?), 985
Poikiloderma atrophicans vascular (Jacobi); parapsoriasis? 987
Sarcoidosis, 983
Urticaria pigmentosa, psoriasis and arthritis, 806
Warts: See Verruca
Wassermann Reaction: See also Syphilis
serologic diagnosis of syphilis; value of complement fixation and agglutination with spirochetal antigens and relation of spirochetal antibody to Wassermann reagin, *455
Waterhouse, L. M.: Case for diagnosis, 987
Waterhouse-Friderichsen Syndrome: See Adrenals, hemorrhage
Way, S. C.: Colloid milium; vitamin deficiency disease (?), *1148
Weber, L. F.: Accidental tattoo, 795
Weber's Disease: See Telangiectasia
Weber-Christian Disease: See Panniculitis
Weber-Dimitri Disease: See Angioma; Brain, calcification
Weber-Rendu-Osler's Disease: See Telangiectasia
Weber-Sturge Syndrome: See Angioma; Epilepsy
Welsh, A. L.: Contact dermatitis from emetine hydrochloride, *550
Werner Syndrome: See Scleroderma
Wetting Agents: See Detergents
Wheal: See Urticaria
White, C.: Alopecia areata of scalp and eyebrows and psoriasis of nails or paronychia congenita, 241
Blastomycosis, 251
White, S. J.: Dermatitis from imitation leather, *1164
White Spot Disease: See Scleroderma
Whitlow: See Paronychia
Wieder, L. M.: Pemphigus erythematodes (Senear-Usher), 994
Wien, M. S.: History of Chicago Dermatological Society, *1138
Wilhelm, L. F. X.: Pyoderma gangraenosum with ulcerative colitis, 177
Winer, L. H.: Fibrotic nodules of skin, *315
Wise, F.: Anetoderma of Jadassohn, 1181
Chronic disseminated lupus erythematosus, 222
Disseminated sarcoidosis, treated with diphtheria toxoid, 1010
Experimental therapy with diphtheria toxoid in 4 cases, 784

Wise, F.—Continued
Granuloma annulare, 818
Leukoderma with inflammatory border, 218
Multiple fibromas; case for diagnosis, 220
Necrobiosis lipoidica, 224
Pili torti; case for diagnosis, 1183
Poikilodermatomyositis, 1009
Tinea due to Trichophyton purpureum or erysipeloid, 1184
Xanthoma diabeticorum with unusual form of eruption; report of case, *723
Xanthoma tuberosum with involvement of palms, 1005

Wolf, C.: Localized myxedema; case for diagnosis, 234

Wolf, W.: Annular papular syphilid, 452
Sensitivity to protamine zinc insulin, *694

Wolfe, H. E.: Pseudoxanthoma elasticum with angioid streaks; syndrome of Groenblad and Strandberg, *1142

Wolfe, L. W.: Solution of aluminum citrate as substitute for solution of aluminum acetate N. F., *514

Wolff, M. J.: Contact dermatitis; case for diagnosis, 188
Erythema annulare centrifugum; case for diagnosis, 190
Herpes zoster associated with generalized varicella-like eruption, 191

Woolhandler, H. W.: Adenoma of sebaceous glands (adenoma sebaceum) with consideration of keratotic adenoma sebaceum and true adenoma of sebaceous glands, *734

Workmen's Compensation and Insurance: See Industrial Diseases

Wounds, treatment of wounds caused by wild animals, 772

Xanthelasma: See Xanthoma

Xanthoma, case for diagnosis, 615
diabeticorum with unusual form of eruption; report of case, *723
or lupus erythematosus with xanthomatous infiltration, 440
tuberosum; hereditary xanthomatosis; familial incidence of xanthoma tuberosum associated with hypercholesteremia and cardiovascular involvement, with report of several cases of sudden death, *1
tuberosum multiplex, 211, 431, 433, 820
tuberosum or xanthelasma, 841
tuberosum with involvement of palms, 1005
xanthomatosis of tendons and bones associated with disease of coronary arteries, 197
xanthomatosis with disease of coronary arteries, 198

Xanthomatosis: See Xanthoma

Xeroderma pigmentosum, 233

Yaws: See Frambesia

Zakon, S. J.: Favus, 237
Necrobiosis lipoidica diabeticorum, 991
Pemphigus erythematodes (Senear-Usher), 791

Zippers, unilateral nickel dermatitis of left forearm caused by zippers, *578

Zoster: See Herpes zoster

Medical Library

JUN 25 1942

VOLUME [illegible] NUMBER 6

ARCHIVES
OF
DERMATOLOGY AND SYPHILOLOGY

EDITORIAL BOARD

HOWARD FOX, Chief Editor, New York

C. C. [illegible], Kansas City, Mo. — FRED D. WEIDMAN, Philadelphia

HAROLD M. [illegible], Cleveland — GEORGE M. MacKEE, New York

HERBERT [illegible], Chicago — C. GUY LANE, Boston

JUNE 1942

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION $[illegible]

Entered as Second Class Matter Oct. 30, 1917, at the Postoffice at Chicago, Under the Act of Congress of March 3, 1879

COPYRIGHT, 1942, BY THE AMERICAN MEDICAL ASSOCIATION

THE A... DERMATOLOGY AND SYPHILOLOGY is published by the Ameri-
can ... sociation to stimulate research and to disseminate knowledge
... in the ... nts of medicine.

Manus... publication, books for review and correspondence relating to
the editorial ... ment should be sent to Dr. Howard Fox, Chief Editor,
1400 East Fif... Street, New York, or to any other member of the Editorial
Board. Com... regarding subscriptions, reprints, etc., should be addressed:
ARCHIVES ... LOGY AND SYPHILOLOGY, American Medical Association,
535 North Dea... Street, Chicago.

Articles ... for publication on condition that they are contributed
solely to the ... OF DERMATOLOGY AND SYPHILOLOGY. Manuscripts must be
typewritten, ... double spaced, and the original copy should be submitted.
Zinc etchings ... used will be supplied by the Association when the original
illustrations ... reproduction and when their number is not considered
excessive.

Footnotes ... bibliographies (the latter are used only in exhaustive reviews
of the literature) ... should conform to the style of the *Quarterly Cumulative Index Medicus*. ... is the order given: name of author, title of article and
name of publ... volume, page, month—day of month if the journal appears
weekly—

Matter ... in THE ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY is
covered by ... but as a rule no objection will be made to its reproduction
in a reputable ... journal if proper credit is given. However, the repro-
duction for ... purposes of articles appearing in the ARCHIVES OF
DERMATOLOGY ... SYPHILOLOGY or in any of the other publications issued by
the Associa... be permitted.

THE ARCHIVES OF DERMATOLOGY AND SYPHILOLOGY is published monthly.
The annual ... price (for two volumes) is as follows: domestic, $8.00;
Canadian, $... ign, $9.00, including postage. Single copies are 85 cents,
postpaid.

Checks, ... ders and drafts should be made payable to the American
Medical Asso...

PERIODICAL PUBLICATIONS
of the American Medical Association

THE ... **AMERICAN MEDICAL ASSOCIATION**—Weekly. Carries all the
official ... items of general medical interest. Illustrated. Annual subscription
price ($2... ; Canadian, $9.50; foreign, $12.00. Single copies, 25 cents.

AMERI... **OF DISEASES OF CHILDREN**—Monthly. Presents pediatrics as
a medical ... a special problem. Includes carefully prepared reviews, based on
recent ... abstracts from foreign and domestic literature, book reviews,
transactions ... ties, etc. Illustrated. Annual subscription price (two volumes):
domestic, ... $8.40; foreign, $8.80. Single copies, 85 cents.

ARCHI... **INTERNAL MEDICINE**—Monthly. Devoted to the publication of advanced
original ... laboratory investigations in internal medicine. Illustrated. Annual
subscription ... volumes): domestic, $8.00; Canadian, $8.40; foreign, $8.80. Single
copies, 75 ...

ARCHIVES OF NEUROLOGY AND PSYCHIATRY—Monthly. A medium for the presentation
of original ... nervous and mental diseases, with abstracts from foreign and
domestic ... reviews, transactions of special societies, etc. Illustrated. Annual
subscription ... volumes): domestic, $8.00; Canadian, $8.40; foreign, $8.80. Single
copies, 75 ...

ARCHIVES OF ...LOGY—Monthly. Devoted largely to the investigative and clinical
phases of ... monthly reviews on orthopedic and urologic surgery. Well illustrated.
Annual ... (two volumes): domestic, $8.00; Canadian, $8.40; foreign, $8.80.
Single copies, ... except special numbers.

ARCHIVES OF OTOLARYNGOLOGY—Monthly. A medium for the presentation of original
articles on ... ear, nose and throat, with abstracts from foreign and domestic
literature, ... transactions of special societies, etc. Illustrated. Annual subscription
price (two ... $8.00; Canadian, $8.40; foreign, $8.80. Single copies, 75 cents.

ARCHIVES OF ...OLOGY—Monthly. A periodical devoted to the publication of
original ... reviews in the field of pathology. Illustrated. Annual subscription
price (two ... domestic, $8.00; Canadian, $8.40; foreign, $8.80. Single copies, 75 cents.
Annual ...

ARCHIVES OF ...NGOLOGY—Monthly. Includes original articles on diseases of
the ... foreign and domestic literature, book reviews, transactions of special
societies, ... Annual subscription price (two volumes): domestic, $8.00;
Canadian, $8.40; ... $9.00. Single copies, 85 cents.

WAR ... Monthly. Devoted to material on preparedness and scientific advance-
ment in ... air, naval, public health and allied services. Annual subscription
price: domestic, $5.00; Canadian, $5.40; foreign, $6.00. Single copies, $1.25.

QUARTERLY CUMULATIVE INDEX MEDICUS—Quarterly. A complete subject and
author ... to the current medical literature of the world. Issued four times a
year, ... volumes bound for permanent reference. Subscription price, calendar
year: ... Canadian, $14.00; foreign, $16.00.

...CAN MEDICAL ASSOCIATION

535 North ... Street CHICAGO

CONTENTS

PAGE

Dermatitis Due to the Blossom of Grevillea Banksii: A Newly Recognized and Common Entity in Hawaii. Harry L. Arnold Jr., M.D., Honolulu, Territory of Hawaii 1037

Dermatitis Due to a Transparent Adhesive Tape. Harry Keil, M.D., New York, and Eugene S. Bereston, M.D., Baltimore 1052

Dermatitis of the Eyelids Due to Philodendron (Scandens Cordatum) Plants. John M. Harris, M.D., New York 1066

Cutaneous Manifestations of Intestinal Malignancy: Types. S. William Becker, M.D., David Kahn, M.D., and Stephen Rothman, M.D., Chicago 1069

Lichen Planochylaris: Lichen Planus et Acuminatus et Atrophicans (Feldman) and Lichen Spinulosus and Pigmentosus Decalvans (Little). Wilhelm Sachs, M.D., Jerrold Cipe, M.D., and Gerard De Oreo, M.D., Cleveland 1081

Electrolysis: Introduction of an Instrument for Relatively Painless Treatment. Eugene A. Hand, M.D., Saginaw, Mich. 1094

Rational Prescription Writing in Dermatology. Herman Goodman, M.D., New York 1101

Dermatophytosis of the Hands: Diagnosis and Treatment. Erwin Epstein, M.D., Oakland, Calif. 1113

Desoxyribonated Pancreatic Extract in Treatment of Psoriasis. John G. Downing, M.D., Earl A. Glickelich, M.D., and Salvatore J. Messina, M.D., Boston 1125

Hemochromatosis: Report of a Case and Consideration of Methods of Making a Definite Diagnosis. Arthur A. Hemmeter, M.D., Sam Alpiner, M.D., and L. E. Verity, M.D., Battle Creek, Mich. 1128

Geographic Distribution of Lupus Erythematosus. Emanuel Gahan, M.D., New York 1133

History of the Chicago Dermatological Society. Max S. Wien, M.D., Chicago 1138

Pseudoxanthoma Elasticum with Angioid Streaks: The Syndrome of Grönblad and Strandberg. Seymour H. Selvin, M.D., Brooklyn, and Herman G. Wolff, M.D., New York 1142

Colloid Milium: A Vitamin Deficiency Disease? Robert C. Wax, M.D., San Francisco 1148

Pigmentation of the Skin Due to Vitamin A Deficiency: Report of a Case and Review of the Literature. Jesse A. Bierbach, M.D., and Thomas N. Graham, M.D., New York 1156

Clinical Notes:

Dermatitis from Imitation Leather. Seymour J. White, M.D., Lawrence, Mass. 1164

Minimal Scarring After Electrodesiccation. Thomas S. Saunders, M.D., Portland, Ore. 1165

Familial Lichen Planus: Report of Twelve Cases in One Family. Marvin R. Goldstein, M.D., Youngstown, Ohio 1166

Special Reports:

First Meeting of the Consulting Staff of the Dermatoses Investigations Section of the United States Public Health Service 1167

News and Comment 1169

Abstracts from Current Literature 1170

Society Transactions:

Manhattan Dermatologic Society 1179

New York Academy of Medicine, Section of Dermatology and Syphilis 1190

Chicago Dermatological Society 1196

Los Angeles Dermatological Society 1210

Book Reviews 1219

General Index 1221